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Symptoms in Relationship to Life Events (Stress, Social Supports, Life Experiences Survey, I-E Locus-Of-Control, Mmpi).

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SYMPTOMS IN RELATIONSHIP

TO LIFE EVENTS

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Abstract

Although there is considerable evidence to support the contributions of life event stress to subsequent psychological and physical symptoms, the correlations reported between obtained stress scores and illness measurements have been low. This study was undertaken to improve prediction of physical and psychological complaints subsequent to life event stress by more comprehensive measurement of life event characteristics and by assessment of locus of control, social supports, and somatization as moderating variables in the life stress-illness process. An overview of psychosomatic medicine was given and the relevant life event research was reviewed. Fifty undergraduate students completed the experimental version of the Life Experiences Survey (LES), Rotter's Internal-External Control of Reinforcement Scale (I-E Scale), the Minnesota Multiphasic Personality Inventory (MMPI), and the Interview Schedule for Social Interaction (ISSI). Subjects then completed psychological and somatic complaint checklists (modified SCL-90-R, modified Wahler Physical Symptoms Inventory) every four weeks for a three month period. Pearson product-moment correlations were calculated, providing an intercorrelation matrix for all

independent and dependent variables, and multiple regression analyses were used to determine the best predictive models. Results did not support the first hypothesis that inclusion of the four idiographic subscales of the experimental LES (desirability, change, anticipation, control) would improve prediction of psychological and/or physiological complaints over the counting of event occurrences. Statistically significant improvement in prediction was found when the measures of social support (AVAT, ADAT%, AVSI, and ADSI scales of the ISSI), locus of control (I-E Scale), and somatization (HS and HY scales of the MMPI) were added to LES scores in an heirarchical regression analysis. However, a more parsimonious and powerful predictive model was derived using a simultaneous stepwise regression analysis: This model consisted of the HS and I-E scales and the change and desirability subscales of the LES. Social support measures were not correlated with either criterion variable nor with any of the predictor variables. These results were discussed in relation to previous studies concerning life event stress, social supports, somatization, and locus of control in relation to health. The clinical implications of this study were also discussed.

INTRODUCTION

Overview of Psychosomatic Medicine

Historical Perspectives

Although psychosomatic medicine as an organized field is only about fifty years old (Lipowski, 1977), it is quite interesting to find that many of the basic themes and issues of today were formulated and recurrently argued over the centuries. The term itself, being derived from the Greek psyche referring to soul, spirit, or mind, and soma meaning body reflects the still controversial mind-body interrelationship that began in prehistoric times. (See Kaplan, 1980, for a more complete review of the mind-body problem as related to medicine historically.)

In primitive society disease was thought to be caused by evil spirits and was fought by spiritual means. Evidence from trepanned Neolithic skulls has suggested that a good many patients of this primitive neurosurgical technique (used to let the evil spirits out) did recover, most likely aided by the suggestive powers of the medicine man, although conceivably through effective reduction of intracranial pressure on occasion. During the Sumerian-Babylonian-Assyrian civilization (circa 2500 to

500 B.C.) medicine was dominated by religion with treatment consisting mainly of prayer, magic, and exorcism; thus Sigerist (1951) concluded that medicine of that time was psychosomatic in all its aspects.

During the Greek and early Roman times (circa 400 B.C. to 400 A.D.) the field of medicine included the most proponents of a holistic view until the recent present. According to Hippocrates (460-370 B.C.), disease was due to imbalance of fluid matter within the body which could be related to or even caused by a similar imbalance in the patient's external environment. He warned doctors to consider such things as wind direction and season of the year before making any diagnosis or beginning treatment. (The field of biometeorology--the study of the ways weather affects living creatures--has begun to be taught in the U.S. in the past twenty years. In West Germany, doctors can dial for a bioprognosis report on how the day's weather may affect health [Ponte, 1982]). As one of the first expounders of holistic medicine, Hippocrates (cited in Dunbar, 1954) wrote that "to cure the human body, it is necessary to have a knowledge of the whole of things." Similarly, consideration of the interaction of the mind and body was expressed by Plato (427-347 B.C.). In Timaeus he stated that trouble in the soul could bring trouble to the body, and he noted the converse in

Charmides by quoting Socrates: "As it is not proper to cure the eyes without the head, nor the head without the body, so neither is it proper to cure the body without the soul" (Plato, 1871). Consideration of emotions and their physiological effects began long ago, with Aristotle (384-322 B.C.) noting that the emotions of fear, anger, courage, and joy affect the functioning of the body. In the First Century A.D. Areteus suggested a disturbance of the emotions as one of the six major causes of paralysis. Galen (130-200 A.D.) reported a case where he considered a differential diagnosis of endogenous versus exogenous depression (i.e., the physical cause of the overbalance of the black bile versus the psychic cause of inordinate desire) and using "behavorial observation", chose the second when he noted an erratic pulse in his female patient at the mention of a dancer, Pylades.

This more holistic approach to medicine and the observation of the interrelationship of mind and body which had developed over the centuries was generally abandoned during the Middle Ages (circa 500 to 1450 A.D.). Mysticism and religion again dominated medicine. Physical and mental diseases were seen as being caused either externally by demons or witches, or internally

through sin or evil within the person. Healing again became a spiritual matter.

During the late Fifteenth to Eighteenth Centuries, with the renewed interest in the natural sciences, the mind/body balance shifted again until eventually the study of the material world dominated. Investigation of the body's structures (e.g., anatomy and autopsy) became the focus of medical science, and the study of the mind was relegated to religion and philosophy. However, advances in physics, mathematics, chemistry, and scientific instrumentation during this period increased the scope of what was accepted as part of the "material" world. Thus, for example, with the development of the microscope, by Leeuwenhoek (1632-1723 A.D.), the theory that matter was composed of tiny particles not visible to the human eye (named atoms by Lucretius in 55 B.C.) now began to be accepted as possible and the study of bacteriology and cellular structure began.

Scientific inquiry continued to expand rapidly, yet the philosophical position proposed by Rene Descartes in the Seventeenth Century--that the mind and body are distinct entities subject to different laws of causality--dominated the study of mental and physical health until recently. Although Gaub (cited in Rather, 1965, p. 71), wrote in 1747 that "the reason why a sound body

becomes ill, or an ailing body recovers, very often lies in the mind," the mind/body schism was greatest in the Nineteenth Century (Kaplan, 1980). Rudolf Virchow (1821-1902 A.D.) determined through laboratory work that the diseased cell was the origin of bodily disease. He showed that the change toward pathological state takes place first within individual cells, second in the cell structure, and then in the tissue of an organ. This somatically-based framework for the study of mind/body functioning was also influenced by scientists such as Thomas Huxley (1825-1895 A.D.) who believed that the mental processes were simply the product of somatic activity, and thus in themselves had no causal significance.

Although scientific investigation of the Nineteenth Century centered on the body, disorders in which emotional/cognitive and somatic activities overlap were recognized. The term psychosomatic disorder was first used by Heinroth in 1818 with regard to insomnia and later popularized by Jacobi in 1822. In 1872, Tuke compiled a vast body of observations and anecdotes in his book, Illustrations of the Influence of the Mind upon the Body in Health and Disease. This seminal work presented a mass of information in a coherent theoretical framework, thus laying the groundwork for a new science. However,

it was not until the early decades of the Twentieth Century that new methods of research and explanatory concepts (e.g., of James, Freud, Pavlov, and Cannon) set the stage for the emergence of psychosomatic medicine from the background of philosophy and medical folklore (Lipowski, 1977).

Psychosomatics from 1900 - 1970

The history of the psychosomatic field in the 1900's reflects movement from the acceptance of an interrelationship of mind and body to a holistic and ecological approach to the field of physical and psychiatric disorders and health. This has been accompanied by fluctuating but overall increasing amounts of interdisciplinary study, research, clinical practice, and training in physical sciences, medicine, allied health sciences, psychology, sociology, and cultural anthropology. From beginnings in the biological and psychosocial disciplines, psychosomatics evolved at the frontier between psychology and medicine. Both clinically and scientifically, the movement fought against a narrow reductionistic medical focus on primarily biological concerns while bringing a "wide spectrum of methods to bear on aspects of human behavior germane to medicine" (Knapp, 1980, p. 1853). This has produced new and overlapping fields of specialization such as psychobiology,

biology, medical psychology, behavioral medicine, neuropsychology, behavioral neurology, and so on.

In the initial development of the field of psychosomatics, however, there were two major directions, psychodynamic and psychophysiological. Overlapping these directions, both in time and in focus, were theoretical trends suggesting that (a) specific conflicts led to specific organ damage (Alexander, 1950); (b) that certain "personalities" were associated with certain psychosomatic diseases (Dunbar, 1954); (c) that nonspecific stress can cause specific physiological changes likely to lead to certain stress diseases or disorders (Selye, 1956); (d) that individuals have certain enduring psychophysiological response patterns (Lacey & Lacey, 1958b; Wolff, 1950); and (e) that stress in general can create preconditions for a number of not necessarily predetermined disorders or even all physical and mental disorders (Mason, 1980). Because of the interwoven nature of these directions and trends, it is easier to follow the development of the field chronologically up until the recent present.

In 1900, Freud noted that somatic involvement occurs in conversion hysteria but limited this concept to organs innervated only by the involuntary neuromuscular or sensory-motor nervous system. He felt that conversion

hysteria represented the symbolic substitutive expression of an unconscious conflict, typically sexual in nature: Hysterical blindness might be due to an unacceptable unconscious wish to be a voyeur (Freud, 1953; Fenichel, 1945). For most of the first thirty years of the century, this essentially psychodynamic focus continued and expanded. The concept of conversion hysteria was applied to organs innervated by the autonomic nervous system. Diseases of these organs (e.g., ulcerative colitis) and/or their symptoms were interpreted as conversion reactions representing perhaps a specific psychic fantasy (Ferenczi, 1926). The influence on the organ tissue of adults by various unresolved pregenital conflicts was proposed by Karl Abraham (1927). Clearly organic conditions, such as fever and hemorrhage, were believed to have primary psychic meanings, representing the expression of unconscious fantasies (Groddock, 1929).

The psychophysiological direction in the field of psychosomatics owes much to the identification of the instinctual fight-or-flight response by Walter Cannon in the 1920's. He noted that a variety of physiological changes occur with stress, the degree and extent of these changes being greatest in a life-threatening situation (Cannon, 1927). However, most of the work in

psychosomatics published before the 1940's continued to have a more psychodynamic emphasis.

The most influential representative of this trend was Franz Alexander who formulated many of the core assumptions of psychosomatic medicine, thus dominating the field from around 1930 to 1955 (Lipowski, 1977). His specificity theory causally linked specific unresolved unconscious conflicts and related emotions in the development of specific disorders, concentrating on seven: bronchial asthma, ulcerative colitis, neurodermatitis, thyrotoxicosis, essential hypertension, rheumatoid arthritis, and duodenal ulcer (Alexander, 1950). He borrowed concepts from Cannon's work, equated conflict with stress, and suggested that the fight-or-flight response could be prolonged with emotional suppression, producing physiological disorders and eventually pathology of the organs of the viscera (Kaplan, 1980). It should be noted that this approach stimulated much clinical research, both of the hypotheses and of the therapy based on them, some of which provided support for his theories (Alexander, French, & Pollock, 1968; Mirksy, 1958). However, this approach had methodological weaknesses, causally linking very different levels of abstraction with poor regard to the intervening physiological mechanisms (Lipowski, 1977). Alexander's theory of

the etiological mechanisms of what he considered to be psychosomatic diseases remains a basically unvalidated hypothesis (Kaplan, 1980) and the efficacy of therapy based on his work did not materialize despite some reported successes (Kellner, 1975).

Like Alexander, Dunbar (1943) concentrated on the seven classical psychosomatic disorders, originally proposing a specific range of conflicts linked to specific theories. This position received little experimental support (Kalucy, 1979). Dunbar (1954) later suggested specific conscious "personality constellations" as causes of specific diseases. Although Dunbar's description of the ambitious, hard-driving man prone to coronary occlusion is similar to the Type A person proposed by Friedman and Rosenman (1974), his position was not generally accepted due to his emphasis on predisposition by personality factors with little or no regard to physiological characteristics (Kaplan, 1980). Others in the psychoanalytic tradition have suggested psychological trauma in birth, infancy, and childhood as predisposing one to adult psychosomatic disease (Deutsch, 1939; Greenacre, 1953).

The psychophysiological direction gained impetus in the 1940's with the studies of Wolff (1950, 1960), with his associate Wolf (1943) and his students Grace and

Graham (1952), linking affective arousal to illness. They demonstrated specific relationships within individuals between conscious affective states and hyper- or hypo-functioning in the vascular and the secretory activities of the musosa of the gastrointestinal and respiratory systems. Although these types of specific changes within individuals were reported to be associated with pathological changes such as bleeding, ulceration, and smooth muscle spasm, Kalucy (1979) notes having found no report of the development of psychosomatic illnesses being explicitly demonstrated as following such pathological changes. Wolff, in addition to his experimental physiological research, also employed epidemiological methods in research on the role of social and psychological factors in disease (Lipowski, 1977).

Mahl (1949) found that with chronic anxiety, gastric hydrocholoric acid production increases. Such acidity is a precursor of peptic ulcer. He concluded that chronic anxiety from whatever source is the variable intervening between the behavioral and physical events in this psychosomatic disease.

Selye (1936) demonstrated that prolonged stress leads to the development of the General Adaption Syndrome (GAS). His earlier work emphasized hormonal changes in the hypophyseal-adrenocortical axis in response to

nonspecific stress, ultimately leading to a variety of organic diseases which he viewed as physical by-products of adaptation to stress (Selye, 1956). Over the years Selye expanded investigations of the physiological bases of adaption. In his later work Selye stated that "the vast majority of all maladies for which the patient seeks medical attention are predominantly due to stress--particularly to psychogenic stress" (Selye, 1980, p. xi).

Another strategy in psychophysiological research in psychosomatics has been the examination of physiological responses within and across individuals to a variety of stimuli. Lacey, Bateman, and Van Lehn (1953), Lacey and Lacey (1958b), and Lacey (1959) are credited with being the first to systematically explore individual differences in patterns of sympathetic nervous system responses, which they called individual response stereotypy. They found that some individuals show maximal activation of the same physiological response pattern across different stimuli and over time. These results would be consistent with a view that an individual whose "natural response" was, for example, to increase gastric secretion rather than systolic blood pressure would be more prone to somatic disorders such as gastritis and duodenal ulcer than to hypertension.

Kalucy (1979) notes a central problem with these physiological studies: It is easier to demonstrate hyperresponsiveness in particular organs or particular reactions to stress than to demonstrate the translation of these to actual disease states.

Beginning in the 1950's and becoming more prominent with time has been the broadening of psychosomatic theoretical perspectives to include the effects of social and ecological factors on disease and health. Grinker (1953) suggested a total approach to psychosomatic disease with multidisciplinary investigators examining ecological, cultural, environmental, emotional, genetic, somatic, and constitutional factors, along with the past history of the individual. Similarly, Weiner, Thayler, Reiser, and Mirsky (1957) proposed a broad field theory, stressing transactional intereffects among biological, psychological, and social factors. More of these factors and more of their interrelationships have been studied since then, with research mainly looking at the relationships between social, psychological, and physiological variables as related to issues of health and illness.

Current Perspectives

A phase of rapprochement between representatives of the fields of psychology and of various medical special-

ties began in the late 1960's (Knapp, 1980); since then psychology has been rapidly assuming a more integral role in health care (Bakal, 1979). In 1974, the 27th World Health Assembly endorsed a holistic, ecological approach to research, practice, and training in medical fields. They urged support of research on the influence of psychosocial factors in health and disease, contending that these factors can both precipitate and protect against physical and emotional illness and are, therefore, critically important in the prevention and management of disease (WHO, 1974). A holistic view has also been endorsed by Weiner (1977) in Psychobiology and Human Disease, wherein he describes disease as a failure of adaptation, a biological phenomena involving organisms in interaction with their natural, social, and cultural environments. According to Lipowski (1977), the current position in psychosomatic medicine advocates an integrative, holistic, and dynamic conception of man as well as of disease. It endorses a doctrine of multicausality of disease, perceiving social and psychological factors as codeterminants of health and illness. It should be noted that the earlier psychophysiological and psychodynamic approaches have not been supplanted, but rather other dimensions have been included in current theory, research, and practice in psychosomatic medicine.

Dominant Theoretical Concepts

The central concerns of psychosomatic medicine are now the identification and study of those factors involved in predisposition to and precipitation of illness, both in general and with regard to specific illnesses, and those variables which enhance resistance to and coping with illness. Such information is needed for the development of social and psychological measures to help prevent or ameliorate the effects of diseases and disorders of all types. Underlying this approach is the core assumption of a gestalt view of human functioning in which physiological processes, overt behavior, symbolic activity, and the environment are all interactive with each other, thus all influencing health and illness. Another basic set of theoretical beliefs involves the role of long-term psychological and physiological tendencies of individuals to react to specific stimulus situations with idiosyncratic patterns of cognitive, behaviorial, emotional, and physiological responses. These responses, if they are maladaptive and prolonged, may act as major precursors or causes of disease. This interactionist model, called the diasthesis-stress model of illness, is reflected in three currently influential areas of investigation: psychosocial stress, psycho-

physiological response specificity, and individual susceptibility to disease.

Psychosocial stress. The concept of psychosocial stress has been complicated by the ambiguity resultant from three meanings for the term stress. It has been used to connote stressors, that is, stimuli that can evoke a response of the organism (Cannon, 1935) or events which challenge the adaptive capacity of the organism (Racy, 1980); a state of the organism characterized by "measurable somatic manifestations, and elicited by a variety of emotional and physical agents" (Selye, 1975, p. 38); or the whole range of interacting factors of stimulus, responses, and the mediating factors (e.g., coping styles, social milieu) which should be viewed together to evaluate the stimulus-response relationship in a specific individual (Lazarus, 1971; Levi, 1974; Mason, 1975). For clarity herein, the term stressor will be used for the stimuli and stress for the totality of the interaction.

Psychosocial stressors are distinguished from other types of stressors (e.g., electric shock) in that the effects are not on the whole directly attributable to qualities of the stressor itself such as intensity and duration. Rather, psychosocial stresses either have acquired the property of eliciting stress responses or,

if innate, do not elicit a stress response based on direct damage to the organism as do physical stressors. Additionally, psychosocial stressors depend more on the mediation of situational and personal factors to determine the nature of the outcome. Situational mediators are the external conditions impinging on the individual (Dohrenwend & Dohrenwend, 1980) such as the availability of financial resources and adequacy of social support systems. Personal mediators are the totality of person factors such as genetic vulnerabilities, effects of past experiences, personality traits or types, attitudes and behaviors. Most current research has focussed on overt and covert behaviors of the individual including symbolic activities such as cognitive appraisal (Lazarus, Cohen, Folkman, Kanner, & Schaefer, 1980), perception of control (Suls & Mullen, 1981), anticipation of the stimulus (Dohrenwend, 1974), subjective meaning (Lipowski, 1977), emotions and their physiological concomitants (Lachman, 1972; Levi, 1975), defensive behaviorial tendencies (Engel & Schmale, 1972), and coping styles (Lazarus, 1982).

Most studies of psychosocial stressors could be classified under one of two major approaches, qualitative and quantitative. Studies using the qualitative approach emphasize certain types of stressors, particularly those

involving loss and/or disaster, which tend to be devastating for most people. Proponents of the qualitative approach such as Engel and Schmale see object loss as a common antecendent to illness (Schmale, 1972). According to this theory, a real, anticipated, or fantasized loss of a valued person, possession or life style can lead to the giving up-given up syndrome, which is associated with helplessness and/or hopelessness and correlated with illness onset. This approach is exemplified by studies of bereavement (Clayton, 1973, 1975; Parkes, 1972; Schmale, 1971), job loss (Kasl, Gore & Cobb, 1975), the bombing blitz of London in 1940-41 (Spicer, Stewart & Winser, 1944), severe injury (Hamburg & Adams, 1967), and prisoners of war (Arthur, 1974). The quantitative approach hypothesizes that life changes are stressors and the magnitude of recent life change is predictive of future illnesses and their severity. The quantitative approach is further subdivided by the issue of whether the stressfulness of life events is idiographic or nomothetic in nature. This question will be discussed further in the section on life events research.

The study of psychosocial stress is further complicated by the fact that it involves a multiplicity of interactive systems. Realistically, researchers can only study a portion of the process examining it as if it were

a one-way sequence. However, it should be emphasized that it is a concept which involves systems internal and external to the individual in which the variables are continuously interacting.

For example, one of the intervening variables between the stressor event and the final outcome is the emotional reactions of the individual. The emotional reactions might have one or more of the following effects:

1. The physiogical concomitants of the emotional reactions could augment, reduce, or change the quality of the emotions. This could lead to changes in the appraisal of the event and thus to further changes in the quality of the emotional reactions and so on.
2. The physiogical concomitants of the emotions could precipitate, exacerbate, or ameliorate a pathological body process. This change in physical functioning could affect the person's emotional state and cognitive functioning which could lead to changes in appraisal and/or coping ability, and so on.
3. The emotional reactions could motivate overt or covert behavior inimical or conducive to well-being. The behavior and its consequences

could also lead to changes in appraisal of or emotional response to the original stressor, and so on.

These three examples do not represent an exhaustive analysis of the possible effects of one of many potential intervening variables; hopefully it does suggest the complexity involved in examining this process.

Psychophysiological Specificity. The concept of psychophysiological specificity relates to the probability that an individual will respond to a specific stimulus situation with a predictable pattern of physiological and psychological changes. This is designated response specificity as psychophysiological specificity is always measured in terms of responses (Roessler & Engel, 1974). If the response is specific to the eliciting stimulus, the term stimulus response specificity (SR specificity) is used. Two concepts are important in examining responses which are specific to the individual rather than to the stimulus. The term individual response stereotypy (IR stereotypy) refers to a heirarchical pattern of responses specific to the individual, occurring in response to a variety of stimuli. The term individual response specificity refers to certain individuals' having a prepotent response system (e.g., cardiovascular), which is most likely to

respond to a variety of stimuli. Although SR and IR specificity are independent in a statistical sense, (Engel, 1972), they are not mutually exclusive. A prediction about a pattern of responses must be based on information about both IR and SR specificity. Additionally, the current psychophysiological state of the person must be taken into account (Lipowski, 1977). In terms of lability of IR sterotypy, individuals range from demonstrating great variability in their response patterns to demonstrating very rigid patterns. Lability as well as pattern of response can be affected by the individual's current emotional state, fatigue, level of autonomic arousal, state of consciousness, and so on.

Investigation of these variables is one prominent area of research into the question of why a particular individual becomes ill at a particular time and why the individual develops the particular illness or maladaptive state he does. In the early 1950's, Wolff (1950) and Grace and Graham (1952) demonstrated relationships specific to individuals between conscious affective states and autonomic functioning. For example, one subject had hyperemic and hypersecretory gastric mucosa when experiencing unexpressed anger and a pale, dry mucosa when depressed. Responses such as these within individuals were noted often to be associated with

pathological changes such as bleeding, ulceration, and smooth muscle spasm. Subsequently, Mirsky (1958) investigating duodenal ulcer postulated IR specificity as one of the etiological factors. Lacey and Lacey (1958a, 1958b) studied psychophysiological response patterns to stimuli within and across individuals over time. They discovered a great variability between subjects in physiological responses to stimuli but found that any one individual tended to demonstrate the same pattern of responses over time (IR stereotypy). Early evidence for stimulus response specificity (SR specificity) was the differentiation of patterns of physiological responses to anger and fear (Ax, 1953; Schacter, 1957) and Lacey's (1959) differentiation between responses associated with sensory intake and with sensory rejection.

Roessler and Engel (1974) note that it often has been impossible to replicate these SR specificity studies and that there has been great difficulty in differentiating physiological response patterns characteristic of other emotions. Roessler (1973) has summarized research indicating that personality or cognitive style contributes a small but pervasive effect upon physiological responsivity. Also it has been noted that emotions are intertwined with the process of cognitive appraisal (Schacter, 1970; Lazarus et al., 1980). Ignoring these

factors has led to research that assumes universal SR specificity (e.g., that naval aviators in combat would all be anxious [Austin, 1969]), thus failing to demonstrate physiological differences which might be present (Roessler and Engel, 1974).

The issues which have been raised in the study of psychophysiological response specificity and the application of this concept in psychosomatic research further illustrate the complexity of investigation in this area. This has led to emphasis of a systems approach to psychosomatic research (Grinker, 1973; Roessler & Engel, 1974) and psychophysiological specificity studies designed to separate out the effects of SR and IR specificity, IR stereotypy, subjective and objective state of the organism, and their interactions (Frankenhaeuser, 1971).

Specificity versus generality. Both specificity and generality theories of psychosomatic etiology accept the contribution of psychosocial variables in the development of illnesses; however, the nature of the link is viewed differently. Although there is great variety within each of these two classes, a general distinction can be made. Specificity theories hypothesize that specific personal characteristics have a predictable relationship to specific physiological variables, diseases, and/or

disorders. Generality theories propose that a wide range of experiences and characteristics may increase general susceptibility to disease or pathological states, thus increasing the probability of illness.

Specificity theories vary greatly in the factors examined and the rigidity of the causal links proposed. Many early specificity theories of etiology were psychoanalytic and linked specific conflicts with specific organ damage (Alexander, 1950), connected certain personalties with specific psychosomatic diseases (Dubar, 1954), or equated psychosomatic illness with conversion reactions (e.g., eczema of the hands as an expression of masturbatory guilt [Allendy, 1932]). Another essentially psychoanalytic approach proposes that a fixation can occur as a function of psychological trauma while the psychological and organ systems are at a certain stage of development. Later situations could then provoke the relevant psychological reaction, simultaneously setting off a train of developmentally linked somatic events, leading to or predisposing one to illness (Engel, 1967).

A more physiological approach to investigating specificity in terms of organ vulnerabilty is based on studies of autonomic conditioning. It has be proposed that specific threatening stimuli may generate a conditioned response in the visceral system mediated by the

autonomic nervous system and leading over time to visceral organ vulnerability (Kalvey, 1979). This type of conditioning as well as genetic factors, past exposure to illness, behavior patterns, cognitive style, coping skills, and numerous other factors have been invoked to account for the individual physiological response patterns discussed previously in the section on physiological response specificity (Lipowski, 1968; Miller, 1975; Whittkower, 1974). A much investigated example of IR stereotypy is the relationship between the Type A Behavior pattern and the development of coronary heart disease and its complications (Friedman & Rosenman, 1974). Recent studies of Type A behavior have introduced the factor of control as an intervening variable affecting the likelihood of this behavioral pattern leading to coronary illness (Frankenhaeuser, 1980). This will be discussed in more detail later.

Generality theories, vary among themselves mainly in their consideration of possible mechanisms linking a wide range of stressors to a subsequently increased likelihood of illness or disorder. The study of the stress process has been approached in terms of physiological, psychological, and psychosocial factors separately and in interaction.

Selye's (1936) early formulation of stress as a set of nonspecific and unitary physiological reactions to various noxious environmental agents was largely responsible for popularizing the concept of stress (Rabkin & Struening, 1976). According to Selye, activation of the pituitary-adrenocortical axis leading to discharge of stress hormones, such as ACTH, adrenal corticoids, and catecholamines, plays a decisive role in the biologic stress syndrome. More recently, Selye stated that essentially the same syndrome is elicited by demands for adaption experienced as positive (eustress) as by noxious adaptive demands (distress). Consequently, he redefined stress as "the nonspecific response of the body to any demand" (Selye, 1975, p. 39). To explain how such a nonspecific, integrated biologic response could lead to different diseases, Selye devised numerous laboratory experiments and concluded that certain <u>conditioning</u> factors determine disease-proneness. He distinguished two types of conditioning factors--internal (e.g., genetic predisposition, age, and sex) and external (e.g., environmental variables, drug usage, and dietary constituents)-- that can selectively inhibit or enhance one or another parameter of the nonspecific stress response (Selye, 1974).

The increased general susceptibility to disease, postulated by other theorists is not seen as a state which occurs in an absolutely unitary nonspecific fashion to all stressors as proposed in Selye's theory (Lipowski, 1977). Mason (1975b) reported that extensive studies in his laboratory on the profiles of multiple hormonal responses did not provide any evidence that "any single hormone responds to all stimuli in absolutely non-specific fashion." Mason suggested that stressors be viewed as analagous to pathogens, that is, as agent which are potentially capable of eliciting specified adaptive responses depending upon certain variables, particularly those pertinent to the individual. He emphasized the susceptibility of different neuroedocrine systems to the effects of emotional arousal in response to various stressors (Mason, 1971).

Neal Miller (1980) includes activation of the pitutary-adrenal axis (CRF release leads to ACTH release leads to corticosteroid release) as one of several neurophysiological mechanisms available for potentially producing a variety of somatic effects of stress. Another possible mechanism he notes is the activation of the sympathetic nervous system causing release of catecholamines and cardiovascular changes among others. He also suggests that pain-inhibiting tracts recently

discovered by Liebeskind and Paul (1977) and the morphine-like endorphins found in these tracts (Snyder, 1979) may have an inhibitory effect on fear and stress from other sources. Studies suggested by Mason (1975) and Miller (1980) on the role of psychological mediators of neuroendoerine response patterns to psychosocial stimuli have been conducted and reviewed by Frankenhaueser (1980). Underlying her research is the belief that the ability of psychosocial variables to arouse the sympathetic-adrenal medullary and pituitary-adrenal cortical systems is dependent on the individual's cognitive appraisal of the balance between the severity of the situational demands and his coping resources. One of the main areas reviewed centers on control as a "key" to coping and thus a determinant of neuroendocrine profile. She summarized research demonstrating that lack of personal control, accompanied by feelings of fear and helplessness, is associated with activation of both the pituitary-adrenal and sympathetic-adrenal systems and that in conditions involving a high degree of control the pituitary-adreanal system may be actively suppressed. These changes in pituitary-adrenal activation and resultant changes in levels of systemic corticosteroids represent an important step in the path from stress to disease since corticosteroids

have immuno-suppressive effects, increasing susceptibility to infection (Miller, 1980). For an excellent review of current knowledge of the immune system and its interaction with the neuroendocrine system, including direct changes in humoral or cellular immunity after natural or experimental stress, see Rogers, Devendra and Reich (1979).

The final major area of generality theories includes studies of life events (also termed life stress and life change) and subsequent illness. These will not be discussed herein as this is the focus of the current project and is elaborated upon in the next section.

Life Event Research

Historical Perspectives

Life events as a factor in the etiology of various diseases has been investigated for approximately twenty years now. This field was first formally recognized at the 1949 Conference on Life Stress and Bodily Disease sponsored by the Association for Research in Nervous and Mental Disease (Rabkin and Struening, 1976). The first large study on the relationship between life events and illness was done by Hinkle and his co-workers in the 1950's (Minter & Kimball, 1980). From their studies of telephone employees over a twenty year period (Hinkle, 1961; Hinkle & Plummer, 1952; Hinkle & Wolff, 1957,

1958), they found that a small percentage of the employees had most of the illness episodes and that they had experienced more stressful situations and events. They hypothesized a temporal causative relationship between stressful life events and the occurrence of illness as well as the presence of inherent differences in adaptive capacities among those who were frequently ill and those who were generally well. Around the same time, Hawkins, Davies and Holmes (1957) noted that 50% of the life events a group of tuberculosis patients reported over a ten year interval clustered in the two years prior to disease onset. Comparing patients who received a diagnosis of tuberculosis with those found to be free of major pulmonary disease, Kissen (1958) observed that the former group had significantly more recent stress events than the latter.

The research on life changes as precipitating stressors in disease became more systematic following Holmes and Rahe's work on standardizing life change measurement techniques (Holmes & Rahe, 1967). Rahe and his colleagues emphasize that recent life changes represent one dimension of life stress and do not typically assess chronic difficulties or anticipated life stressors. However, they feel that all changes require adjustment, and that the psychological and physiological

efforts involved in such adjustment, if severe and/or protracted, may predispose individuals toward developing illness (Rahe and Arthur, 1978). Scores on the first version, known as the Schedule of Recent Experience (SRE) were based on the number of events experienced. Subsequently, weights were assigned to each event based on ratings by a standardization sample of judges. These judges were asked to rate each life event as to the relative degree of necessary readjustment in terms of the intensity and time needed to accommodate to the specific life event. This weighted scale, known as the Social Readjustment Rating Scale (SRRS) has been used in the original form or with modifications for specific populations (e.g., children, college students, and athletes) by the majority of investigators working in this research field (Rabkin & Struening, 1976).

Using these scales and others, life event stress has been found to be related to complications of pregnancy and parturition (Gorsuch & Key, 1974), sudden cardiac death (Rahe & Lind, 1971), heart disease (Hinkle, 1974; Holmes & Masudia, 1974; Thorell, 1974), diabetes mellitus (Stein & Charles, 1971) rheumatoid arthritis (Heisel, 1972), seriousness of chronic illness (Wyler, Masudia, & Holmes, 1971), as well as numerous other specific health problems (Rabkin & Streuning, 1976). A systematic

analysis of over 300 published studies between 1965 and 1975 notes "illness in general" as the third condition most studied, with cardiovascular-renal first and cancer second (Hull, 1977). In addition to its relationship to physical illness, life change stress has been found to correlate with involvement in serious traffic accidents (Selzer & Vinokur, 1974; Sobel & Underhill, 1974), academic (Harris, 1972) and work performance (Carranza, 1972), injuries to football players (Branwell, Masuda, Wagner, & Holmes, 1975), psychiatric symptomatology (Dekker & Webb, 1974; Paykel, 1974), onset of acute schizophrenia (Birley & Brown, 1970), suicide (Paykel, 1976), anxiety and depression (Vinokur & Selzer, 1975), and other types of psychiatric disorders (Brown, 1974; Hudgens, 1974). These studies and numerous others (see reviews by Dohrenwend & Dohrenwend, 1978; Minter and Kimball, 1980; Rabkin & Streuning, 1976; Rahe & Arthur, 1978) support Hinkle's conclusion that the individual's relation to his psychosocial environment affects all aspects of human growth, development, and disease (Hinkle, 1974).

Current Issues and Directions

Measurement of Life Stress

Four major issues have arisen in the measurement of stressful life events. First, what defines a life event

as stressful -- change or undesirability? Second, should events be weighted in terms of "stressfulness, " however defined, or simply counted in terms of the summed numbers of event occurrences? The third question is whether idiographic or nomothetic measurement of stressful events is more appropriate. Finally, other characteristics of the events or event-related moderator variables, such as anticipation, control, and novelty, have been postulated to be important.

The question of what constitutes the general class of stressful life events has produced some controversy. Holmes and Rahe, in their ground breaking 1967 study, defined stressful life events as those "whose advent is either indicative of or requires a significant change in the ongoing life pattern of the individual" (p. 217). Most early investigations used one of the Holmes and Rahe scales (SRE and SRRS) thus, tacitly at least, accepting this definition. Currently, it is still generally accepted that stressful life events involve change in the usual activities of most individuals who experience them. However, many investigators have further refined the definition of stressful life events to distinguish those that are likely to be experienced as negative from those experienced as positive or neutral (Dohrenwend and Dohrenwend, 1980). The SRE and SRRS are based on the

assumption that life changes per se are stressful and therefore desirable, undesirable, and neutral events are combined in determining the life stress score. Several researchers have questioned the logic of this combining of events, arguing that undesirable events may have a more detrimental effect on individuals than positive events (Brown, 1974; Mechanic, 1975; Sarason, DeMonchaux & Hunt, 1975). Recent studies (Johnson & Sarason, 1978; Mueller, Edwards, & Yarvis, 1977, 1978) have suggested that life stress is related essentially to the number of negative or undesirable events, and that desirable events contribute little to the prediction of subjective strain (McFarlane, Norman, Streiner, Roy & Scott, 1980). Similarly, Vinokur and Selzer (1975, p. 344) concluded that the contribution of life events to psychological impairment relates to "some undesirable aspect of the events rather than change per se."

However, a number of studies suggest that both positive and negative life changes contribute to physical illness (Coppel, 1980; Holmes & Masuda, 1974; Petrich & Holmes, 1977; Rahe & Arthur, 1978; Sarason, Levine, & Basham, 1980). Sarason, Levine, and Sarason (1982) suggest that perhaps negative changes are associated with psychological distress while life changes in general stress the body's physiological homeostasis as previously

hypothesized by Holmes and Rahe (1967). Another possibility is that many positive life events may necessitate changes in one's daily routine which are less desirable. For example, a job promotion which provides increased salary and status may also involve long working hours, more time pressure, loss of old working relationships, and so on.

After examining twenty-three different methods of weighting life events to predict symptomatology, Ross and Mirowsky (1979) concluded that the best undesirability score predicts symptomatology better than change scores. Two studies have investigated the relationship between the dimensions of change and undesirability. One study (Paykel & Uhlenhuth, 1972) showed that 8 of 19 events were rated significantly differently on these two dimensions. Another study (Tennant & Andrews, 1976) found a correlation of .44 between ratings of 66 events on the dimension of change and emotional distress. Dohrenwend and Dohrenwend (1980) conclude that these two dimensions cannot be substituted one for the other even though they are not completely independent.

It has been questioned whether weighting events provides additional information over counting of life events. Two studies comparing the SRE and the SRRS (weighted form), found that counting of events correlated

highly (.97 and .93) with scores using differential weights (Hurst, Jenkins & Rose, 1978; Lei & Skinner, 1980). Antonovsky (1974) found that weights provided no additional information as compared to the summed number of life events. In a survey of weighting schemes by Ross and Mirowsky (1979), it was concluded that the most predictive and efficient undesirability index consisted of the summed number of undesirable events. However, they did not analyze any idiographic weighting schemes. Pertinent to this observation is the finding of Hurst et al. (1978) that a simple count is not an equivalent substitute for assessment of the self-rated impact life events had on individuals (idiographic weighting).

Whether assessment of the impact of stressful life events is important may vary depending on whether one uses a nomothetic or an idiographic weighting scale. All the studies which found no difference between weighted and simple summed scores assessed nomothetic scales. These scales (e.g., Holmes & Rahe, 1967; Paykel, Prusoff, & Uhlenhuth, 1971) predict the impact of events on individuals from average or "judged" perceptions of events. Those who support an idiographic conception of stressful life events (e.g., Breznitz, 1980; Frankenhauser, 1980; Hinkle, 1973; Lazarus, 1980; Rahe, 1974; Thorell, 1974, Vinokur & Selzer, 1975) assert that

cognitive processes, particularly the individual meaning assigned to an event in light of one's unique life situation, should be considered in assessing the impact of that life event. For example, the event of pregnancy is quite different when an unmarried adolescent of a strict moral family accidently becomes pregnant and a happily married woman who has been trying to have a child becomes pregnant. These two experiences would produce different levels of stress, differ greatly in terms of desirability, and also in terms of the amount of subsequent change in the life pattern of each individual.

This example is also pertinent to two other characteristics of life events which have been suggested to influence their impact, that is, the degree to which an event is anticipated and/or controlled by the individual (Dohrenwend & Dohrenwend, 1974; McFarlane, et al., 1980; Pennebaker, Burnan, Schaeffer, & Harker, 1977). Reviews of stress as studied under laboratory conditions (Averill, 1973; Lefcourt, 1973; Miller, 1980) have also suggested that anticipation and control affect the impact of noxious stimiuli or situations. In discussing the psychological aspects of life stress, Frankenhaueser (1980) reviewed research on the role of personal control as a mediator of neuroendocrine response patterns. The results suggested that in stressful situations where

there was lack of control accompanied by feelings of fear and helplessness catecholamine and cortisol levels increased, but under conditions of high controllability and/or predictability cortisol secretion might be actively suppressed. This research, thus, indicates a possible neuroendocrine mechanism underlying the finding of reduced stress impact in laboratory studies allowing personal control (Miller, 1980), in life events studies for events over which individuals felt they had control (McFarlane et al., 1980), and in studies of stressful psychosocial conditions when control is a factor (Frankenhaueser, 1980). One study (Suls & Mullen, 1981) has suggested that uncertainty about one's potential for control may be more devastating than lack of control. They found a significant life events-illness relationship for undesirable, uncontrolled events ($\underline{r}$ = .23) but an even stronger relationship ($\underline{r}$ = .47) for undesirable events of uncertain controllability.

Another characteristic postulated to be important in modifying the impact of events is novelty. It has been suggested (Dohrenwend & Dohrenwend, 1978) that repetition will bring habituation and thus reduce or eliminate the stress response to an event. Similarly, it has been postulated that lack of prior experience heightens the impact of stressful life events (Rabkin & Struening,

1976). Although this may well be true for some events and for some persons, it would seem that there are several confounding factors that argue against inclusion of this characteristic in measurement of life events currently. Dohrenwend and Dohrenwend (1978) themselves note one likely exception in terms of event timing. They state that an undesirable event repeated in fairly rapid succession would probably lead to amplification rather than reduction of stressfulness of an event. However there are several factors they do not consider. It is likely that what would constitute "fairly rapid succession" would vary dependent on the type of events. Experiencing several changes in social activities over a short period of time (e.g., three months) could easily result in reduced experience of stress related to this particular event, whereas more than one death of a close friend or a family member over a much longer period of time (e.g., one to two years) would more likely result in augmentation of stress related to this particular event.

Additionally, the cognitive appraisal of events interacts with the effects of event repetition and nature of event. Coyne and Lazarus (1980) provide a comprehensive discussion of the complex interaction of cognitive style, stress perception, and coping. Their model would suggest that the effects of event repetition depend

greatly on an individual's appraisal of his or her coping resources. Persons who had coped well with a similar event previously would likely feel competent to cope currently and appraise the event as irrelevant or benign (e.g., not stressful). If, however, individuals believe they coped poorly previously or that their coping resources are now depleted, they may perceive the event as threatening where it otherwise would not be.

The phenomenon of habituation, according to laboratory studies, suggest that given the proper temporal arrangement unconsequented stimulus repetition would result in some diminution of the response to the stimulus. However, life events rarely have no consequence. Thus, the phenomenon of habituation is less applicable to this type of stimuli. The likelihood of habituation is also decreased by the fact that life events occur contextually and are never the exact stimulus constellation experienced previously in contrast to laboratory studies of this phenomena. A thorough investigation of behavioral studies and research on the physiological substrates of habituation revealed no research on this phenomenon in relation to event occurrence in natural settings (Peeke & Herz, 1973-1976).

These four major issues involved in the measurement of life stress exemplify the complexity of the assessment

of the events themselves and suggest important considerations for any life event research instrument. It would appear that an adequate instrument should (a) ascertain the contribution of negative and positive events both numerically and with an idiographic weighting system, (b) assess undesirability of as well as change consequent to an event, and (c) examine other event-related characteristics of stressful events which have been demonstrated to influence impact, such as the extent to which events were anticipated and/or were under personal control. Other event-related characteristics such as event novelty may be important in terms of event impact, but have not been adequately investigated yet and may be too complex to be included in life event measurement currently.

Moderating Factors, Person-Related

In the previous section on measurement of life stress, it was noted that certain event-related factors (e.g., anticipation of the event) influence the ultimate impact of the event. In addition, event impact has been found to be moderated by certain characteristics of the person experiencing the event. Although several authors (Dohrenwend & Dohrenwend, 1978; Jenkins, 1979; Johnson & Sarason, 1979; Rabkin & Streuning, 1976; Rahe & Arthur, 1976) have pointed to the important role of moderator

variables, most studies have been designed to simply assess the role of stressful life events in contributing to the onset of physical illness or emotional malfunction (e.g., depression). Sarason et al. (1982) opine that lack of attention to moderator variables has constituted a major limitation of much of the life change research. In their survey of life event research, Rabkin and Streuning (1976) noted that the correlations reported between obtained stress scores and illness measurements in the studies they examined ranged from .28 to .78 with the majority being below .30. It has been hypothesized that as moderators of stress are identified and then measured within life event research designs, effectiveness in prediction is likely to increase.

In their model of the life stress process, Dohrenwend and Dohrenwend (1980) conceptualize two broad categories of moderators: external and internal. External moderators are environmental conditions which impinge on the individual and affect the nature of the outcome of psychosocial stress. They include material and social supports or handicaps, such as ethnic status, income, social support systems, and nutrition. Internal moderators are characteristics of the person that arise from psychological or physical attributes such as

biological vulnerabilities, cognitive style, coping strategies, and intellectual abilities.

It has been suggested that physiological moderators relate more to the kind of symptoms or illness (e.g., cardiovascular versus gastrointestinal) an individual is likely to have than to the time of onset (McFarlane et al., 1980). However, rate of onset is likely influenced by certain physiological variables such as response system baseline and reactivity. Psychological characteristics are important to the evaluation of events as irrelevant, benign, or stressful, and to the appraisal of one's current coping resources, options, and constraints (Coyne & Lazarus, 1980). Psychological characteristics are, therefore, likely to moderate the relationship between occurrence of events and their impact as demonstrated by onset of physical or emotional symptomatology and/or illness.

External factors. Several external moderating factors have been investigated such as memberships in socially marginal groups (Linsky, 1970), status or role incongruence (Cobb & Kasl, 1966), occupational prestiege (Lin, Ensel, Simeone, & Kuo, 1979), social integration (Myers, Lindenthal, & Pepper, 1975), sex (Dohrenwend & Dohrenwend, 1976; Gove & Tudor, 1973), marital status (Bachrach, 1975; Pearlin & Johnson, 1977), and rural

versus urban location (Dohrenwend & Dohrenwend, 1974b; Kasl & Harburg, 1975). However, social support has been the factor predominating the research recently.

The social support hypothesis essentially argues that individuals experiencing stressful life conditions will be less adversely affected if they have a good social support system and conversely, that lack of social support has a negative effect on physical and psychological health. Social support has been included as a factor in most stress-adaptation models (e.g., Antonovsky, 1974; Cobb, 1974; Dohrenwend & Dohrenwend, 1980; Mechanic & Volkhart, 1974; Moss, 1973).

The inclusion of social supports as a factor in models and theories initially tended to be based on studies in which social supports were not directly measured or on evidence that was inferential or indirectly related to the hypothesis (e.g., Brown & Harris, 1978; Cassel, 1976; Cobb, 1976; Dean & Lin, 1977). However, over the past few years, several empirical studies have provided more direct support for the effect of this factor in stress and illness. Social supports have been found to be a significant factor in health outcomes including complications of pregnancy (Nuckolls, Cassel, & Kaplan, 1972), need for steroid medications in chronic intrinsic asthma (De Araujo, Van Arsdel, Holmes, &

Dudley, 1973), illnesses following job loss (Gore, 1978), psychiatric symptoms (Lin et al., 1979), neurosis (Henderson, 1981), psychological well-being (Burke & Weir, 1977), psychological and physical symptomatology (Miller & Ingham, 1976), recovery following myocardial infarction (Finlayson, 1976), subjective strain (McFarlane et al., 1980) and mortality (Berkman & Syme, 1979).

Although research on the effects of social support variables on stress and illness continues to proliferate, the area of study is still in its infancy. Despite the general agreement that social supports do play a role in the stress-illness process, there is a lack of concensus as to what exactly constitutes a social support and therefore, much inconsistency in measurement of the factor. Additionally, the function or role of social supports within the stress-illness process needs further investigation.

Two definitions of social support illustrate some of the differences that have arisen. Lin et al. (1979) define social support as the support accessible to an individual through social ties to other individuals, groups, and the larger community. Sarason et al.(1982) define social support as the existence or availability of people on whom we can rely and who let us know that they care about, value, and love us. Both definitions are

broad yet have very different emphasis, reflecting the lack of concensus as to the nature of social supports. Schaefer, Coyne, and Lazarus (1981) have pointed out that some of the most frequently cited studies treat social network, psychosocial assets, and perceived social support as interchangeable concepts, thus limiting the integration of existing findings as well as the generalizations that can be made about the effects of social support.

One important distinction is between social network and perceived social support. The social network has been defined (Mitchell, 1969) as the specific set of linkages among a particular group of persons, or alternately as the set of relationships of a given individual. The network can be described or measured in terms of size, structure or density (the number of people involved who know each other), context of relationships (e. g., work, social, religious) or by the content of particular relationships (e.g., friendships versus kinship). However, so far the only major study of social networks and health outcomes (Berkman & Syme, 1979) used essentially a measure of size--the Social Network Index--consisting of marital status, number of close friend and relatives, and membership in church and other groups.

This index predicted all-cause mortality rates in a large adult population over a 9.5 year period.

Using this index, the population was divided into four categories reflecting differences in type and extent of social contact, from least to most connected. They used a chi square modification which allows more than two comparison groups, with the statistic adjusted for up to two covariates. The relative risk of those most isolated to those with most connections, ranged from 1.8 to 4.6, with the age-adjusted relative risk being 2.3 for men and 2.8 for women. The age-adjusted differences in mortality among the four categories was highly significant ($p < .001$). To eliminate the possibility of these difference being attributable to other causes, several factors were used as covariates along with age, and a statistically significant gradient was still found for each covariate as follows: baseline health status (p .001), socioeconomic status ($p < .001$), smoking ($p < .001$), obesity ($p < .001$), alcohol consumption ($p < .001$), physical activity (p .001), cumulative health practice index ($p < .001$), and health service utilization ($p < .005$). This research, although impressive in scope and methodology, was limited by measuring only network size.

The use of size measures to infer the benefits of social relationships makes two questionable assumptions:

(a) that benefits are proportional to the size and range of the network and (b) that having a relationship is equivalent to getting support. These assumptions ignore the demands, constraints, conflicts (Schaefer et al., 1981), and nonsupportiveness (Gore, 1981) that are also associated with social relationships. These potential "costs" of social ties have only been specifically investigated in terms of the family as a source of stress (Croog, 1970) and as a factor in the etiology of psychopathology (Liem & Liem, 1978).

The quality of relationship is considered in the concept of perceived social support. Perceived social support involves evaluation or appraisal of whether and to what extent an interaction, relationship, or pattern of interactions or relationships is helpful (Schaefer, et al., 1981). A similar distinction between the structural (network) and evaluative components of support has been made by Henderson (1981) in his investigation of availability versus perceived adequacy of social ties and their relationship to adverse events and neurosis.

Studies which have used size of social network (Berkman & Syme, 1979) or availability of close relationships and diffuse ties (Miller & Ingham, 1976) have found a postive relationship between their assessment of social support and physical and psychological health

outcomes. However, recent studies (Andrews, Tennant, Hewson, & Vaillant, 1978; Chan, 1977; Henderson, 1977, 1981; Porritt, 1979; Schaefer et al., 1981; Wilcox, 1981) have suggested that it is the quality of the supporting emotional relationships rather than the quantity of support available that determines the effectiveness of social supports in dealing with adversity or crisis.

In addition to the distinction between availability and adequacy of social supports, various functions of social support have been suggested as being relevant to the stress-health relationship. Dean and Lin (1977) distinguished two functions, expressive (i.e., emotional or affiliative) and instrumental (e.g., material aid, information), but regarded the expressive as more important. Noting that virtually all studies of social support have emphasized the expressive function over the instrumental, Schaefer et al. (1981) investigated three types of support: emotional, tangible, and informational. Their results suggested that all three types of support were positively associated with positive morale and that both tangible and emotional support were inversely associated with depression and negative morale; none were correlated with physical health status. Other studies have also suggested the importance of

availability of instrumental support during stressful life events (Carveth & Gottlieb, 1977; Kaplan, Cassel, & Gore, 1977).

The lack of consensus as to what constitutes social support is reflected in the way it has been assessed as well as how it has been defined and the functions emphasized or investigated. Assessment has ranged from brief questionnaires, such as a six item scale measuring one's perception of the extent to which the individual has access to emotional support systems (Fleming, Baum, Gisriel, & Gatchel, 1982) to elaborate schemes for mapping social networks (e.g., Phillips & Fischer, 1981).

Almost all studies have used instruments or measures developed for that particular study and give little or no data on test construction, reliability, standardization, and validity (e.g., Andrews, et al., 1978; Berkman & Syme, 1979; Billings & Moos, 1982; Miller & Ingham, 1976; Nuckolls et al., 1972; Porritt, 1981).

In the social support literature, another question has been raised: At what point(s) in the stress-illness process do social supports have an effect? The three main hypotheses which have been addressed are the stress preventative, the stress-buffering (or alternately the stress-vulnerability), and the direct health benefits hypotheses.

The first perspective, stress preventative, suggests the possibility that on-going social support activity might determine vulnerability to the occurrence of those stress events over which individuals have some personal control. This position suggests that support could reduce the likelihood of illness by reducing the likelihood of negative events occurring. For example, a supportive friend could provide suggestions about increasing a person's work productivity and thus prevent the individual from being fired. Evidence for this perspective would be in terms of direct main effects on level of stress by level of support. This possible function has not been the focus of systematic research activity (Gore, 1981). However, one study (Lin et al., 1979) which did investigate this possibility found no support for it.

The second perspective, which has been discussed both in terms of vulnerability and buffering, suggests that the presence of social supports are effective in reducing or buffering the impact of life stress (Dean & Lin, 1977) and conversely, a lack of social support could be conceived as a determinate of increased vulnerability to the effects of stressful events (Kessler, 1979). Social support in this viewpoint has been considered as serving a coping function (Gore, 1978), enabling one to cope better with a stressful event through obtaining

emotional support, information, advice, material aid, and so forth. The consequence of seeking supports could be to affect the relationship between the objective stressful event and the subjective perception of stress or between the subjective stress and the outcome or impact measures (Gore, 1981). Similar possibilities are suggested by Pearlin and Schooler's (1978, p. 2) discussion of two ways in which coping behaviors can serve a protective function following a stressful event: "by perceptually controlling the meaning of experience in a manner that neutralizes its problematic character, and by keeping the emotional consequences of problems with manageable bounds." In this moderating role, social support could also be a resource for provision of information and material aids and service (instrumental function). For some stressful events, support system members could provide all these stress-buffering forms of support. For example, one life event mentioned in most scales is a change of residences. Members of a good support system could act to lessen the stress of moving by helping the person who is moving focus on the positive aspects of the change, empathizing with his or her feelings about moving, providing information about the new neighborhood, helping the person pack, lending a truck to use in moving, and so on.

In examining social supports as moderating the impact of stress, some differences between the stress-buffering and vulnerability hypotheses emerge. Most proponents of the stress-buffering position hold that high levels of support aid people in coping with stress, but in the absence of significant stress, level of support is neither beneficial nor detrimental to physical and psychological well-being. Several studies have shown the types of interactions between stress and support which would suggest that having a high level of social support is beneficial primarily when stress is relatively high (Andrews, et al., 1978; Billings & Moos, 1982; Caplan & Killilea, 1976; Dean & Lin, 1977; DeAraujo et al., 1973; Gore, 1978; Henderson, 1981; Kaplan et al., 1977; Meyers, et al., 1975; Nuckolls, 1972). The vulnerability position would suggest that in addition to high levels of stress being mitigated by high levels of social support, low levels of stress would have significantly greater impact on individuals with low support than with high support. Two studies (Frydman, 1981; Lin et al., 1979) have provided some evidence for this position; however, their findings were inconclusive as the differences were not found at all levels of all combinations of support and symptomatology variables used in either study.

The third perspective on the effect of social support in the life stress process, which herein is termed the direct health benefit position, holds that having little or no social support is detrimental and a high level of support is beneficial to physical and psychological health regardless of the presence of stressful events or conditions (Fleming et al., 1982). Research has indicated that general benefits such as longer life, better health, and lower morbidity rates are associated with high levels of support (Berkman, 1977; Berkman & Symes, 1979; Cassel, 1976). Other studies have found a significant relationship between social support variables and health status in the absence of a significant stress-support interaction (Andrew et al., 1978; Gore, 1978; Liem & Liem, 1975; Miller & Ingham, 1976; Schaefer et al., 1981). These findings have been interpreted to support the position that social support has a direct and additive effect with life stress on illness rather than a moderating effect. However, these interpretations have been qualified by some of the investigators who made them. Gore (1981) suggested that the distinction between statistical patterns of main and interaction effects may be somewhat artificial in this context and is "overly rigorous in light of the state of the methodological sophistication in the field" (p. 204).

Frydman (1981) discusses the questionable appropriateness of looking for the types of interaction effects assessed by conventional ANOVA or regression analysis with multiplicative terms in this area of research, having found direct and conditional (moderating) effects without any interaction effects. Additionally, Schaefer et al. (1981) note that it would be premature to conclude that social support has no buffering effect, suggesting that these effects may be more evident if support were assessed with respect to the demands of specific stressful occurrences.

The somewhat conflicting and inconclusive evidence presented in favor of different effects of social support is actually not very surprising considering the lack of concensus as to what constitutes social support (social network versus perceived social support), what functions of social support should be measured (expressive versus instrumental), and the consequent variety of often poorly researched instruments used in the assessment of social support. Given this state of affairs, the fact that researchers do consistently find social support effects of some type suggests that it is an important variable to be considered in stress and illness research. The weight of evidence supports a buffering hypothesis at this point. However, it is quite possible that social support

also affects the occurrence of stressful events, moderates the impact of stressful events, and additionally directly benefits well-being. Pragmatically, from the point of view of those who are interested clinically in mental and physical health and illness, the question as to how this variable is related to life events (protective, additive, or interactive) is not all that important (Day, 1981). Better assessment of this factor, however, will be useful in the design of primary and secondary intervention strategies. In terms of intervention, social support is a factor which is potentially accessible and more susceptible to manipulation unlike stressful life events themselves which tend to be extremely difficult to manipulate experimentally and difficult or, sometimes, impossible to reverse (Day, 1981; Lin et al., 1979).

A final issue which should be considered is the possible confounding of effects due to overlap in measurement of stressful life events and social supports (Gore, 1981; Schaefer et al., 1981). Life events lists include items which represent actual reductions in the size of a person's social network (e.g., bereavement) or potential reduction in the amount, availability, or quality of social support (e.g., new job, increase in arguments with spouse,a move to a different part of a city). Thus, loss events would be assessed twice, once

in terms of increase in the stress variable (life event) and second as a decrease in social support availability. One study (Schaefer et al., 1981) has specifically addressed this issue and found that loss-type events were inversely associated with social support and social network size, although only a few of the correlations were statistically significant. However, when the effects of recent losses were controlled, there were still significant correlations of social support with depression and morale, indicating that the effects of low levels of perceived support were independent from those of loss of social ties. A surprising finding was that loss-type life events were not associated with depression, negative morale, or decline in physical health. Schaefer and her associates suggest that this finding might have been due to the fact that there were many fewer loss events than nonloss events for their subjects. Typically, the prevalence of major life events such as bereavement or divorce has been found to be low in most subject populations studied (Goldberg & Comstock, 1980).

The foregoing discussion has emphasized the diversity of opinion and evidence as to what exactly constitutes social support, what are the important functions of a good support system, and how support systems affect the stress-illness process. Additionally, the possibility of

confounding of stress and social support due to inclusion of social loss-type events in life event lists has been considered. Given the current lack of concensus in the literature, it would seem that future studies of the stress-related functions of social support should assess this factor in terms of availability (network size) and adequacy (perceived social support) and consider instrumental as well as expressive functions.

Internal factors. In the attempt to increase prediction effectiveness for pathological outcomes related to stressful life events, several internal moderating factors related to individual differences in temperament, beliefs, and behaviors have been investigated. A wide variety of internal factors have been investigated including assertiveness (Tanck & Robbins, 1979), hardiness (Kobasa, 1979), Type A behavior pattern (Friedman, & Rosenman, 1974), social conformity, liberal intellectualism, and emotional sensitivity (Garrity, Somes, & Marx, 1977), sensation-seeking (Cohen, 1982; Cooley and Keesey, 1981a; Johnson, Sarason & Siegel, 1979; Smith, Johnson, & Sarason, 1978), tendency to develop psychophysiological disorders (Cooley and Keesey, 1981b), anomie (Jenkins, 1979) and locus of control (Johnson & Sarason, 1978; Lefcourt, 1981; Linn, Linn, &

Harris, 1981; McFarlane et al., 1980; Tanck & Robbins, 1974).

Although some evidence has been found for the utility of including any of the above factors in studying the stress-illness process, two factors have particular significance in terms of psychotherapeutic intervention: locus of control and tendency to develop psychophysiological disorders. An integral part of the typical psychotherapeutic process is development of an increased sense of responsibility for and control over one's behavior and its effect on the environment--which is closely related, if not identical to the concept of locus of control (Hill & Bale, 1981). Locus of control is a concept proposed by Rotter (1962, 1966, 1975) that individuals have a generalized expectancy concerning the extent to which the rewards, punishments, and, in general, the events in their lives are contingent upon their own behavior or relatively permanent characteristics. Rotter conceived expectancy as varying from internal to external. Persons who are extremely internal expect to be in control of what happens in their lives to a high degree, whereas those who are extremely external expect that what happens in their lives will generally he controlled by others or by fate. Internal locus of control has been empirically associated with more

adaptive coping with stress (Tank & Robbins, 1974) and capacity for reducing stress effects cognitively (Ferguson, 1979); external control has been shown to be associated with greater likelihood of psychological and/or physical illness symptoms following stress (Johnson & Sarason, 1981; Kobasa, 1979; Lefcourt, 1981; Linn et al., 1981). Thus, the locus of control factor is an important addition to the study of the stress-illness process for two reasons: (a) its inclusion should increase the power to predict likelihood of illness following life event stress, and (b) if further empirical evidence substantiates external locus of control as a factor in vulnerability to illness, there already are strategies for intervention through psychotherapeutic modification of individuals' control expectancies.

The second factor which has relevance in terms of intervention is the tendency of some individuals to development of psychophysiological disorders in response to stress. A classic MMPI interpretive pattern, the conversion vee, which is formed by elevations (T Score 65) on scales 1 and 3 (Hypochondriasis and Hysteria), accompanied by a lower score on scale 2 (Depression) has been empirically associated with psychophysiologic reactions, especially under stressful conditions (Dahlstrom, Welsh, & Dahlstrom, 1972; Gilberstadt &

Duker, 1965; Graham, 1977). This tendency to develop physical symptoms consequent to psychological or psychosocial stressors is also known as somatization. Given the wide use of the MMPI clinically, it is surprising what little attention has been paid to this somatization factor as a possible moderating variable in the stress-illness process. A survey of relevant literature over the past five years uncovered one study which examined this variable. Cooley and Keesey (1981) compared two groups, those subjects who experienced change while showing fewer illnesses than would be expected (coping subjects), and those who showed more illness that their level of life change would have predicted (sensitive subjects). The MMPI results showed scales 1 and 3 to be significantly higher for the sensitive group than for the coping group with the K scale and scale 2 showing no differences. (Only these four scales were administered in this study.) This MMPI pattern is typically associated with a coping style which includes a tendency to deny psychological problems and lack insight into one's own emotional life (Dahlstrom et al., 1972; Gilberstadt & Duker, 1965; Graham, 1977). It has been suggested that hypochondriasis and psychosomatic symptoms are the most common "masks" of depression in adults in the United States (Lesse, 1980). They are called masks

as they serve to cover over underlying psychological distress. Thus, it seems that persons with this type of coping style would be more likely to respond to stress with physical symptoms or disorders. Some confirmation for this hypothesis can be found in the work of Glass (1977) on persons with the Type A behavior pattern, which is associated with proneness to the development of heart disease. His results have suggested that the Type A pattern may involve repression of responses to threatening stimuli. In both of the above behavioral patterns, the more immediate response to stress (typically a perception of emotional distress) is pushed out of awareness, thus making activation of appropriate coping responses less likely, and the end result of physical illness more likely since the stressor still exists to exert its influence on physiological processes. This type of behavioral pattern seems likely to be a factor in vulnerability to physical illness subsequent to stress and needs further investigation. Additionally, if this does prove a vulnerability factor, there are various psychotherapeutic strategies available for intervention in this process. More traditional psychotherapy typically has as one of its aims increased insight into one's behavior and the bringing into awareness of perceptions or feelings which have been repressed. More behaviorally

oriented techniques, such as stress management and biofeedbeack therapy, can help individuals become aware of the cognitive, emotional, and physical cues of distress, decrease physiological reactivity to stress, and learn more appropriate cognitive and behavioral coping strategies.

The role of moderating variables in life event research has been reviewed with emphasis on three factors; social support, locus of control, and somatization. All three factors have received empirical support as having a role in the stress-illness process. The social support factor has been the most widely researched with much support for its playing a role in the stress-illness process, although there is little consensus yet as to definition, conceptualization, and technique of measurement for this factor. Locus of control is a well-defined concept with strongly established measurement techniques. In the life event studies including this factor, the evidence has supported external locus of control as an illness vulnerability factor. Somatization has been extensively studied in general but included in only one life event study thus far. That study did support this as a likely factor in vulnerability to physical symptoms or disorders. Inclusion of moderator variables such as these should

increase ability to predict symptoms and/or illness subsequent to life event stress. The three emphasized herein, moreover, are factors amenable to intervention. It has been noted that modern life is inherently stressful (Brodsky, 1977; Toffler, 1970) and that it is difficult and sometimes impossible to prevent or reverse the occurrence of stressful life events (Day, 1981; Lin et.al., 1979). However, future research can help decrease vulnerability to the effects of stress through identification of moderating factors which are amenable to social and psychological intervention.

Illness Measures

The early work in life event research by Rahe and his colleagues emphasized physical illness as the dependent measure. Rahe in his 1975 review reported various measures of physical illness, such as number of illnesses reported to medical personnel during one year at sea, survey of Navy health records, occurrence of medical illnesses or injuries which prevented men from completing underwater demolition training. Based on a survey of life event research with general population groups published in the past five years, the emphasis has shifted. Currently, there is a fairly even division between use of physical and psychological complaints as the illness measure. Examination of the dependent

measure specified in the twenty-eight studies surveyed revealed that twelve used measures of physical complaints, eleven used measures of psychological complaints only, and five used measures of both. (It should be noted that there are also numerous studies examining the life event stress-illness process in particular patient groups, both medical and psychiatric, which were not included in this survey.)

Further examination of these studies reveals a variety of measures used to assess both physical and psychological complaints. For a complete listing of specific measures used, see Table 1. Of the 15 studies using one or more measures of physical complaints, five used or included the Seriousness of Illness Rating Scale (SIRS) developed by Wyler, Masuda and Holmes (1968); seven used or included a variety of briefly described nonstandaradized checklists or questionnaires assessing occurrence of illness and/or symptoms, two used an unspecified measure of illness occurrence, one included a physician's rating of general health status, one used a questionnaire followed by a standardized medical case-taking interview, one used comprehensive documentation contact along with a health diary kept for three days out of every two weeks, one used assay of urinary catecholamine levels, and one study assessed viral infection

Table 1

Survey of Illness Measures in Life Events Research

Author(s)	Type	Measure	Time
Andrews et al., 1978	PSYC	General Health Questionnaire (GHQ)	R, N-0
Billings & Moos, 1982	PHYS	Checklist of 12 Symptoms	R
Cooley, et al., 1979	PHYS	Seriousness of Illness Rating Scale (SIRS)	R
Cooley & Keesey, 1981a	PHYS	SIRS, modified	R
Cooley & Keesey, 1981b	PHYS	Not specified	R
Fleming et al., 1982	PSYC	Beck Depression Inventory	C
	PSYC	SCL-90	
	PHYS	Urinary catecholamine levels	
Frydman, 1981	PSYC	General Well-Being Scale	R, N-0
	PSYC	GHQ	
Garrity et al., 1977	PSYC	Langner's psychiatric impairment scale	P
	PHYC	Reports of health problems	
Garrity et al., 1978	PHYS	SIRS, modified	P
	PHYS	Reports of health problems	
	PHYS	Subjective estimate of overall health status	
Henderson, 1981	PHYC	GHQ	P
Jenkins, 1979	PSYC	Psychiatric Status Schedule	P

(table continues)

Author(s)	Type	Measure	Time
Johnson & Sarason, 1978	PSYC	Beck Depression Inventory State-Trait Anxiety Inventory	C
Kanner et al., 1981	PSYC	Hopkins Symptom Checklist (HSC) Bradburn Morale Scale (BMS)	C & P
Kobasa, 1979	PHYS	SIRS	R
Lefcourt, 1981	PSYC	Profile of Mood States	P
Lin et al., 1979	PSYC	Checklist of 24 symptoms	R
Linn et al., 1981	PSYC	HSC	R
McFarlane et al., 1980	PSYC	Langner's Scale	P
	PHYS	Health diaries	
	PHYS	Physician contract reports	
Murphy & Brown, 1980	PHYS	Health questionnaire Standardized medical case-taking interview	R & C
	PSYC	Present State Examination Interview (short version)	
Otto, 1979	PHYS	Checklist of 20 symptoms	R
	PHYS	Physician contacts	
Pennebaker et al., 1977	BOTH	Pennebaker Inventory of Limbic Languidness (PILL)	
Schaefer et al., 1981	PHYS	Health Status Questionnaire	P
	PSYC	HSC	
	PSYC	BMS	
Smith et al., 1978	PSYC	Psychiatric Screening Inventory	C
Stone & Neale, 1981	PHYS	SIRS	R

(table continues)

Author(s)	Type	Measure	Time
Suls & Mullen, 1981	PHYS	Report of illness	R
Tessler & Mechanic, 1978	PHYS	Report of illness	R & C
	PHYS	Perceived health status	
	PHYS	Physician health rating	
Totman et al., 1979	PHYS	Presence of rhinovirus, ten days post-innoculation	P
Yunik, 1980	PHYS	Checklist of health problems	P

Note: Time refers to the time period which the illness measures covered with respect to the initial data collection. P = prospective; R = retrospective; C = current; that is, symptoms or illnesses presnet at time of initial data collection. (Life events were measured retro- spectively.) PHYS = Physical; PSYC = psychological.

following experimental nasal inoculation with rhinoviruses. Psychological measurements included a variety of standardized psychological instruments as well as psychiatric questionnaires and structured psychiatric interviews using a specified standardized format. See Table 1 for further details.

In addition to the variety of methods used for assessing physical and psychological complaints, varying time orientations and time periods were used. Thirteen studies assessed complaints retrospectively for periods of one to twelve months prior to the date on which they collected all of the data. A few of these attempted to separate the life event and the complaint measurement periods (e.g., complaints for the past month, life events for previous 2-13 months), but for most retrospective studies the measurement period for these two variables was concurrent or overlapping. Five studies used or included a measurement of health or psychological status at the time of data collection. Nine studies assessed complaints, doctors visits, and/or psychological status prospectively, with one or more assessments occurring from one month to two years following the original data collection. One experimental study (Totman, Kiff, Reed & Craig, 1979) measured the amount of virus present daily

for 10 days following inoculation with rhinovirus. (The time orientation and periods are included in Table 1.)

It is generally agreed that prospective measurement of complaints is preferable where possible and that self-reports of symptoms, illnesses, and doctor visits are more reliable the shorter the reporting period. Self-report of physical complaints has been used in most studies for several reasons. Pragmatically, it is often difficult to obtain access to all the medical records of any given subject group. Additionally, studies have found a high degree of correlation between conditions reported by subjects and those found in their medical records, and between self-health perception and medical assessments, suggesting that self-report information concerning health status is reliable (Linn & Linn, 1980; Maddox & Douglas, 1973; Suchman, Phillips, & Streib, 1958). It has also been questioned whether using doctor's visits as a dependent measure is relevant due to variability in reasons for visits (e.g., care for continuing versus acute conditions), infrequent use of laboratory tests to assess complaints presented to physicians (resulting in subjective, albeit professional, determination of the complaint being organic versus functional), and the resultant lack of distinction between illness and illness behavior (Ingham & Miller,

1978; Thurlow, 1971). The trend away from use of doctor visits as a dependent measure and to use of self-report emphasizing symptoms rather than illnesses is exemplified in the more recent writings of both Holmes and Rahe, the pioneers of life event research. In a recent interview, Holmes (1982, p. 70) stated that "life change does not--and I emphasize not correlate with . . . visits to the doctor. It has nothing to do with what's called 'seeking patient care' or seeking treatment procedures." Rahe and Arthur (1978) have noted that although recent life changes remain a significant predictor of near-future illness, much stronger associations are seen between recent life changes and subsequent levels of psychological and physiological symptoms.

This summary statement by Rahe and Arthur also underlines the trend to use of psychological complaints as a dependent measure, with physical and psychological complaints being measured about equally as often in the current literature. Two recent studies (Cooley & Keesy, 1981a; Scheafer et. al., 1981) have suggested that psychological measures may be more sensitive to the effects of life change than physical illness. Murphy and Brown (1980) found the relationship between life events and onset of organic illness to be mediated by an intervening affective disturbance. Thus far, only a few

studies have used both psychological and physical complaint measures which would allow for exploration of such possibilities.

Specific Goals of the Present Investigation

The purpose of the present investigation was two-fold: (a) to attempt to improve prediction of physical and psychological complaints subsequent to life event stress by more comprehensive measurement of life event characteristics, and (b) to further improve prediction of physical and psychological complaints by including assessment of moderating factors in the stress-illness process. It was hoped that this would also illuminate the relationships between physical and psychological complaints, illness occurrence, and the tendency to develop psychophysiological disorders within the life event research framework.

A review of the literature on measurement of life stress suggested that using nomothetic weighting of events does not increase prediction of subsequent illness over simple counting of event occurrence but that idiographic weighting may improve prediction. Past research has suggested that certain characteristics of the events may affect their impact and therefore by assessing these event characteristics, prediction would

be improved. It has been suggested that life event measurement should include assessment of the perceived undesirability of an event as well as the change consequent to the event, and measurement of the extent to which events were anticipated and perceived to be controllable.

Hypothesis 1: It was predicted that by including idiographic assessment of life event characteristics (i.e., change, desirability, anticipation, and controllability), stronger correlation with subsequent illness and/or complaints would be found than with simple counting of event occurrence.

One of the current emphases in life event research has been on the identification of moderating factors to improve prediction in the stress-illness process. Many factors have been investigated already out of the multitude of possibilities. The present research included three factors which have received empirical support as mediating variables in the stress-illness process. The factors chosen were ones that are also amenable to change with existing psychotherapeutic strategies, thus having relevance for intervention in the stress-illness process.

Hypothesis 2: It was hypothesized that by including assessment of social supports, locus of control, and somatization as moderating variables, stronger correlations between life event stress and subsequent complaints would be obtained.

METHODOLOGY

Subjects

The subjects consisted of fifty undergraduates from an introductory level psychology class at LSU in Baton Rouge, Louisiana. The sample consisted of 28 females and 22 males. The students ranged in age from 17 to 35 years with a mean age of 19.5 years. There were 4 married and 46 single subjects, with 46 being white and 4 nonwhite. Of the married subjects, 3 were females and one was male. It had been hoped that there would be at least 30 subjects of each sex with an equal number of married males and married females included. This was not feasible due to the high level of attrition during the various stages of the study. A minimum of 50 volunteers appeared to provide the degrees of freedom needed for statistical analysis with allowance for subject attrition (D. Blouin, personal communication, July 19, 1983).

Volunteers were solicited during a psychology class following a lecture on stress. A form was distributed to the students requesting their participation and asking them to provide information about their sex, marital status, address, phone number, and availability for a subsequent testing session (see Figure 1). At this initial point, 159 students volunteered to participate and completed the first measure. (Full description of

Figure 1. Form used for solicitation and voluntary consent of subjects.

The Psychology Department is conducting a research project to investigate the relationship between the occurrence of life events and subsequent physical and/or psychological complaints. Your participation is voluntary and any information that you provide will be kept totally confidential. Participation consists of taking three paper and pencil tests in a group administration (approximately 1½ hours), answering questions in a 45 minute interview, and turning in an answer sheet for two complaint checklists (approximately 15 minutes) three times (every four weeks) before the end of the semester. You are free to terminate your participation at any time. All information obtained about any individual will be kept confidential. If you would like a copy of the study results, mark the box next to your address.

A random sample of subjects will be drawn from those people who agree to participate. If you would be willing to participate, please fill in the following information, and sign this form.

I understand the requirements for participation in this research project and would be willing to participate.

Sex: Male _____ Female _____ Marital Status: Married _____ Unmarried _____

Mailing Address: _____

_____

Phone Number: _____

Please indicate any and all of the following times that would be convenient for your participation in the group administration testing.

Monday,	10 am	_____	3 pm	_____	5 pm	_____	7 pm	_____
Tuesday,	3 pm	_____	5 pm	_____	7 pm	_____		
Wednesday,	10 am	_____	1 pm	_____	5 pm	_____		
Thursday,	10 am	_____	3 pm	_____	5 pm	_____	7 pm	_____

the stages of the study are included in the procedure section of this chapter.) After analyzing the times students were available based on the solicitation form, six group session times for the following week were determined and sign-up sheets posted before and after the next class session; the sheets were then posted for the remainder of that week on a bulletin board reserved for solicitation of volunteers for psychology experiments in Audubon Hall at LSU. A seventh group session was added, announced, and posted in the same way to allow any students who were unable to come the week of the original six group sessions, and who still wished to participate, to do so. For this second stage, 73 students signed up and 67 participated. Sixty students participated in the third stage, and of those 56 correctly completed and turned in all the checklists for the dependent variables. The data from 6 of these 56 students were not included in the final analysis as their MMPI F-K scores did not meet validity criteria. All subjects who completed all phases of this study received class credit for participation.

Materials

The subjects' experiences of life events along with event-related moderator variables of desirability, change, anticipation, and control were assessed using a revised version of the Life Experiences Survey (LES).[1]

Availability and adequacy of social support were assessed with the Interview Schedule for Social Interaction (ISSI).[2] Locus of control was measured using the Internal-External Control of Reinforcement Scale (I-E Scale). Somatization was assessed using the Hysteria and Hypochondriasis scales of the Minnesota Multiphasic Personality Inventory (MMPI). Psychological symptoms were measured with the modified SCL-90 and physical symptoms with the modified Wahler Physical Symptoms Inventory. A complete description of these instruments, including their reliability, validity, and discussion of previous research in which they were used, are found in Appendices I - VI.

Procedure

The independent variables included the Life Experiences Survey (LES), Rotter's I-E Scale, the Interview Schedule for Social Interaction (ISSI), and the MMPI. The LES was self-administered during a psychology class period as part of a lecture on the topic of stress. At that time, this research project was explained and those who volunteered to participate in the study were

given a choice of six group administration times during which they completed the I-E Scale and the MMPI. The ISSI was administered individually in a face-to-face interview lasting approximately 45 minutes. The ISSI interviews were completed prior to examination of the LES, I-E Scale, and MMPI data to avoid the possibility of any contamination due to examiner bias.

The dependent variables included a physical complaint measure, the Wahler Physical Symptom Inventory (WPSI), and a psychological complaint measure, the SCL-90-R. At the time of the group administration of the MMPI and the I-E Scale, the directions for responding to the SCL-90 and the WPSI were explained and any questions answered. The subjects were given a copy of each instrument and three score sheets with instructions to fill out one score sheet at the end of each four week interval for the twelve weeks subsequent to administration of the LES. The date due was noted on each answer sheet. The score sheet included space for two responses to each SCL-90-R and WPSI item on one sheet (see Figures 2 and 3). For this study, subjects were instructed to respond as to whether and how often the item occurred in the past two weeks, based on a five point scale (0 = never to 5 = almost daily). Then, if it occurred, they also responded as to how much it had

bothered them on a separate five point scale (0 = not at all to 5 = extremely). For the purpose of statistical analysis, a combined score representing the sum of the ratings for all items on both scales (bother and occurrence) was derived for the SCL-90-R and for the WPSI. The same time frame (two weeks) and the same scoring system for both dependent measures was used to facilitate comparision of physical and psychological complaints. Score sheets were turned in at subjects' scheduled class period or at the Psychology Department office. Students were reminded to complete and turn in the score sheets during their class periods the week prior to each due date. Any subjects who did not turn in their forms within two days of the due date were contacted by the experimenter as a further reminder.

Analysis

Data analysis consisted of six multiple regression analyses using the General Linear Model Procedure of the Statistical Analysis System (Ray, 1982). Due to the differences noted in the Introduction between occurrence and/or time of onset of physical and psychological symptoms subsequent to life event experiences, it seemed likely that the best set of predictors might not be the same for both dependent variables. Therefore, separate multiple regression analyses were run for each criterion

Figure 2. Answer sheet instructions for the SCL-90-R and the WPSI questionnaires.

Instructions: Before reading any further, be sure you are using the correct answer sheet for the current due date. For the lists of problems and complaints on the SCL-90-R and the WPSI, read each item carefully and mark your response on the separate answer sheet provided. The first two sets of columns (items numbered 1-90) provide spaces to respond to the SCL-90-R and the second set of columns for responses to the WPSI (items numbered 1-42). Do not mark the questionnaires. For each item, respond first as to whether and how often that item occurred in the past two weeks, using the the Occurrence Scale ratings below. Mark your occurrence rating in the O column on the answer sheet. If that item did not occur, go to the next item. If that item did occur, then rate how much you were bothered by it using the Bother Scale ratings below. Mark your bother rating in the B column on the answer sheet. Be sure to use the correct columns for each test and check to make sure the test item number matches your response sheet number. You must respond to all items--DO NOT SKIP ANY! Please print your number clearly. If you need to change a response, erase carefully and clearly mark your new response.

Occurrence Scale (O)	Bother Scale (B)
0 = Never	0 = Not at all
1 = Rarely (once)	2 = A little bit
2 = Occasionally (2-3 times)	3 = Moderately
3 = Frequently (6-10 times)	4 = Quite a bit
4 = Almost daily (11+)	5 = Extremely

Example 1. Item a. -- body aches (WPSI)
Example 2. Item a. -- nervousness (SCL-90)

Answer Sheet	SCL-90-R		WPSI	
	O	B	O	B
a.	2	3	0	_

These answers indicate that the repondent experienced "nervousness" occasionally (O rating of 2) in the past two weeks and it bothered him moderately (B rating of 3). He did not experience body aches at all (O rating of 0), and therefore, made no response in the B column. Note: Whenever you put a "O" in the O column, leave B blank.

Figure 3. Answer sheet for the SCL-90-R and WPSI Items.

I.D. No. _____ Date Due: _____

SCL-90-R

	O	B		O	B
1.			46.		
2.			47.		
3.			48.		
4.			49.		
5.			50.		
6.			51.		
7.			52.		
8.			53.		
9.			54.		
10.			55.		
11.			56.		
12.			57.		
13.			58.		
14.			59.		
15.			60.		
16.			61.		
17.			62.		
18.			63.		
19.			64.		
20.			65.		
21.			66.		
22.			67.		
23.			68.		
24.			69.		
25.			70.		
26.			71.		
27.			72.		
28.			73.		
29.			74.		
30.			75.		
31.			76.		
32.			77.		
33.			78.		
34.			79.		
35.			80.		
36.			81.		
37.			82.		
38.			83.		
39.			84.		
40.			85.		
41.			86.		
42.			87.		
43.			88.		
44.			89.		
45.			90.		

WPSI

	O	B
1.		
2.		
3.		
4.		
5.		
6.		
7.		
8.		
9.		
10.		
11.		
12.		
13.		
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42.		

variable: (a) physical symptoms as measured by the WPSI combined score and (b) psychological symptoms as measured by the SCL-90-R combined score. For the independent variable of life event occurrence, five scales obtained from the LES were used: (a) occurrence, (b) desirability, (c) change, (d) anticipation, and (e) control. For the independent variable, locus of control, the score from Rotter's I-E Scale was used. For the independent variable of somatization, the Hy and Hs scales of the MMPI were used. For the independent variable, social support, the ISSI AVAT, ADAT%, AVSI, and ADSI scales were used. To test the first hypothesis, the "occurrence" subscale of the LES was forced into the first position in the multiple regression equation and the four other (idiographic) life event subscales were allowed to enter in a nonhierarchical stepwise fashion in separate analyses for each criterion variable. To test the second hypothesis, the best model obtained from the test of the first hypothesis for each criterion variable was forced into the initial position in the next multiple regression analysis for each criterion variable, and the subscales of the independent variables were allowed to enter in a nonhierarchical stepwise fashion. Simultaneous or nonhierarchical stepwise multiple regression equations were also derived using all of the predictor subsales for each criterion variable to explore

the possibility that a more powerful and/or parsimonious set of predictor variables could be derived.

Experimental Hypotheses

1. It was hypothesized that the ability of the LES to predict future psychological and/or physical complaints would be enhanced by inclusion of measures of the event-related moderating variables of desirability, change, anticipation, and control. These variables were measured using the revised LES (experimental version) which includes four separate scales for assessment of these specific factors. Psychological complaints were measured with the modified SCL-90-R and physical complaints were assessed with the modified WPSI. If the addition of these scales did improve prediction, then the amount of variance accounted for when these special scales were entered in to derive a regression equation as compared to the variance accounted for by only the basic score (total number of event occurrences) would reflect the degree to which this hypothesis was supported. F-tests were done to determine if the regression models had been improved significantly.

2. It was hypothesized that the ability of the LES to predict future physical and/or psychological complaints would be enhanced by the inclusion of measures reflecting the person-related moderator variables of

social support (the AVAT, ADAT%, AVSI, and ADSI scales of the ISSI), locus of control (Rotter's I-E Scale), and Somatization (the Hy and Hs scales of the MMPI). Subsequent to entering the LES scores, these seven scale scores were entered into the regression equations for prediction of the physical and psycholgoical complaint scores (obtained from the WPSI and the SCL-90-R, respectively). It was hypothesized that if addition of these scales did improve prediction, then the increase in variance accounted for by entering these scales into the equations would reflect the degree to which this hypothesis was supported. F-tests were done to determine if the regression model had been significantly improved by adding this group of moderator variables.

RESULTS

The major findings of the study will be examined first in relationship to the two experimental hypotheses. This will encompass the results of four stepwise regression procedures, one for each of the two dependent (criterion) variables for each of the two hypotheses. In order to support or reject a specific hypothesis, a procedure was used which entered one or more of the independent (predictor) variables into the regression equation in a predetermined order, allowing comparison of different predictive models. Since the above procedure is essentially hierarchical, the resultant proportion of variance attached to, and thus, accounted for, by each predictor or independent variable is order-dependent. Thus, to determine whether a model with a more powerful or more parsimonious set of predictor variables could be derived without regard to an experimental hypothesis, a simultaneous stepwise regression procedure was also utilized. The models derived using this procedure for each dependent variable will be presented and compared with the models which resulted from the procedures used to assess the experimental hypotheses. The descriptive statistics for and intercorrelations among the variables will also be presented and discussed.

Hypothesis I

The first hypothesis of the study, that inclusion of idiographic measures of life event characteristics would result in stronger correlations with subsequent psychological and/or physical complaints than found with simple counting of event occurrence, was not supported for either of the two criterion variables: psychological complaints as measured by the SCL-90-R combined score and physical complaints as assessed by the WPSI. In order to test this hypothesis, a regression procedure was used in which the occurrence subscale of the LES (LIFE 1) was forced into the regression equation first and then the other four subscales were simultaneously correlated with and regressed on the criterion variable. As can be seen from Table 2, the independent variable LIFE 1 was significantly correlated with psychological complaints, $\underline{R} = .3890$, $\underline{R}^2 = .1513$, $\underline{F}\ (1,48) = 8.56$, $\underline{p} < .0052$. Although the addition of the four idiographic life event measures (LIFE 2 = desirability, LIFE 3 = change, LIFE 4 = anticipation, and LIFE 5 = control) to the measure of life event occurrence (LIFE 1) resulted in an increase of the correlation coefficient from .3890 to .4219, this did not represent a significant improvement in predictive ability, $F\ (4,44) = 0.36$, n.s., using the F-test for extra sum of squares (Neter & Wasserman, 1974). LIFE 1 was also significantly correlated with

Table 2

Regression Analysis of Life Event Factors on Psychological Complaints, LIFE 1 Forced

INCLUDED VARIABLE ENTERED — R SQUARE = 0.15133667

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	1	64013.20468551	64019.20468551	8.56	0.0052
ERROR	48	359012.87087004	7479.26697646		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	61.21000979				
LIFE1	6.96344637	3.06372686	64019.20468551	8.56	0.0052

VARIABLE LIFE4 ENTERED — R SQUARE = 0.18344216

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	2	69140.13332451	34570.06661246	4.59	0.0151
ERROR	47	353884.94223064	7529.46685810		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	54.68294293				
LIFE1	13.99151249	6.62799583	31616.01005829	4.20	0.0461
LIFE4	-1.69912196	2.3038.328	5120.93883940	0.68	0.4137

MODEL IS THE BEST 2 VARIABLE MODEL FOUND.

VARIABLE LIFE3 ENTERED — R SQUARE = 0.17562658

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	3	74379.25213914	24793.01737971	3.27	0.0294
ERROR	46	348645.82341642	7579.26137862		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	66.36214735				
LIFE1	9.63442831	13.58432278	735.94675111	0.10	0.7567
LIFE3	2.74843132	3.39540696	5238.91891422	0.69	0.4100
LIFE4	-1.97229305	2.31121100	5515.13526430	0.73	0.3981

MODEL IS THE BEST 3 VARIABLE MODEL FOUND.

VARIABLE LIFE5 ENTERED — R SQUARE = 0.17630869

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	4	74532.99557491	18645.74896873	2.41	0.0632
ERROR	45	348492.07956065	7743.12732624		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	67.31845772				
LIFE1	9.20642368	13.12532134	347.97710966	0.04	0.8331
LIFE3	2.69349090	3.38216674	5434.45968664	0.70	0.4066
LIFE4	-2.03745165	2.46093552	5624.77512033	0.73	0.3986
LIFE5	0.44875719	2.76512824	283.94373577	0.03	0.8718

MODEL IS THE BEST 4 VARIABLE MODEL FOUND.

VARIABLE LIFE2 ENTERED — R SQUARE = 0.17800256

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	75299.54750772	15059.90950154	1.91	0.1127
ERROR	44	347775.52804783	7902.85291018		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	65.97482149				
LIFE1	6.27511609	13.36715514	322.45170785	0.12	0.7342
LIFE2	-2.79624484	9.20631476	716.55163231	0.09	0.7647
LIFE3	2.76920023	3.42351969	5170.67161886	0.65	0.4229
LIFE4	-1.05525729	2.49012672	5393.64217751	0.68	0.4136
LIFE5	1.05152972	3.43669111	739.85396155	0.09	0.7611

MODEL IS THE BEST 5 VARIABLE MODEL FOUND.

Note: In all steps of this procedure LIFE 1 was included as the first variable, i.e., forced into the first position in each model. All other independent variables were entered in a simultaneous stepwise fashion. LIFE 1 = desirability; LIFE 3 = change; LIFE 4 = anticipation; LIFE 5 = control.

physical complaints $\underline{R}$ = .4916, $\underline{R}^2$ = .2417, $\underline{F}$ (1,48) = 15.30, $\underline{p} < .0003$. For this second criterion variable also, the best five variable model had a correlation, $\underline{R}$ = .5421, $\underline{R}^2$ = .2938, which was not significantly higher than that based on event occurrence alone, $\underline{F}$ (4,44) = 0.81, n.s. See Table 3 for a detailed presentation of these analyses.

Hypothesis II

The second hypothesis, that inclusion of three moderator variables which assessed social support, locus of control, and somatization would result in stronger correlations between life event stress as measured by the LES and subsequent complaints, was supported for both criterion variables, (psychological and physical complaints). To assess this hypothesis, the LES five subscale model derived in the first analysis for each criterion variable was forced into the regression equation first and the seven subscales of the moderator variables were then simultaneously correlated with and regressed on each dependent variable in two separate analyses. Examination of the data for the criterion variable psychological complaints presented in Table 4 demonstrates that the addition of all the scales of the three moderator variables resulted in a twelve variable model with a correlation coefficient of .7138 as compared to .4219 for the five variable model LIFE 1 - LIFE 5.

Table 3

Regression Analysis of Life Event Factors on Physical Complaints, LIFE 1 Forced

INCLUDED VARIABLE ENTERED — R SQUARE = 0.2410?707

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	1	20743.38762001	20743.08762001	15.30	0.0003
ERROR	48	65079.58126888	1355.82460977		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	1.23756934				
LIFE1	5.10216954	1.30443260	20743.08762001	15.30	0.0003

VARIABLE LIFE3 ENTERED — R SQUARE = 0.2b631114

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	2	24571.93631551	12285.99315776	9.43	0.0004
ERROR	47	61250.6025?33b	1303.20601220		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	11.446683414				
LIFE1	-3.33960603	1.12163767	573.84867103	0.44	0.5102
LIFE3	2.34793536	1.35979547	3828.39869550	2.94	0.0931

MODEL IS THE BEST 2 VARIABLE MODEL FOUND.

VARIABLE LIFE4 ENTERED — R SQUARE = 0.2b935400

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	3	24613.132?1305	8277.71080435	6.24	0.0012
ERROR	46	61209.53647584	1325.85948861		
TOTAL	49	85822.66688889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	10.07335972				
LIFE1	-2.36650128	1.64355956	226.07183861	0.17	0.6816
LIFE3	2.37129306	1.39265173	3859.03023831	2.94	0.0931
LIFE4	-0.42917571	0.96703482	261.14609753	0.20	0.6593

MODEL IS THE BEST 3 VARIABLE MODEL FOUND.

VARIABLE LIFE2 ENTERED — R SQUARE = 0.2?3290?1

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	4	25170.13?71059	6292.53323025	4.67	0.0031
ERROR	45	60652.53596792	1347.83413262		
TOTAL	49	85822.66688889			

	P VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	11.13156800				
LIFE1	-4.82?24376	7.57915244	546.75609620	0.41	0.5274
LIFE2	1.55974263	3.11729111	337.02050794	0.25	0.6195
LIFE3	2.47075504	1.40?01?192	4149.?1306105	3.08	0.0861
LIFE4	-0.5464?024	1.30280817	430.16404931	0.30	0.5885

MODEL IS THE BEST 4 VARIABLE MODEL FOUND.

VARIABLE LIFE5 ENTERED — R SQUARE = 0.293a3496

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	25217.70349224	5043.54069845	3.66	0.0074
ERROR	44	60604.96039665	1377.38564538		
TOTAL	49	85822.66688889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	11.22473377				
LIFE1	-4.770260?3	7.66792261	533.07001466	0.39	0.5371
LIFE2	1.13509180	3.97685204	116.90895079	0.09	0.7703
LIFE3	2.49?98853	1.42?25150	4135.65931320	3.35	0.0879
LIFE4	-0.58921014	1.33957213	442.43038870	0.32	0.5737
LIFE5	0.?6662695	1.43475231	47.56757125	0.03	0.8534

MODEL IS THE BEST 5 VARIABLE MODEL FOUND.

Note: In all steps of this procedure LIFE 1 was included as the first variable, i.e., forced into the first position in each model. All other independent variables were entered in a simultaneous stepwise fashion. LIFE 1 = occurrence; LIFE 2 = desirability; LIFE 3 = change; LIFE 4 = anticipation; LIFE 5 = control.

Table 4

Regression Analysis of Life Event Factors, Locus of Control, Somatization Factors, and Social Support Factors on Psychological Complaints, LIFE 1 - LIFE 5 Forced.

INCLUDED VARIABLES ENTERED R SQUARE = 0.17802256

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	75547.5x750772	15059.9[illegible]0154	1.91	0.1127
ERROR	44	347738.52b04783	7902.85251018		
TOTAL	49	423285.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	65.974[illegible]149				
LIFE1	6.27511639	18.36715514	932.45170765	0.12	0.7342
LIFE2	-2.79624464	9.28631[illegible]7E	716.55163281	0.09	0.7647
LIFE3	5.76920323	3.42351969	3170.67161686	0.65	0.4229
LIFE4	-2.02325725	6.49011872	4361.[illegible]2177[illegible]	0.60	0.4136
LIFE5	1.35152972	3.43669111	739.85596195	0.09	0.7611

VARIABLE HS ENTERED R SQUARE = 0.[illegible]1931

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	6	1[illegible]0.51[illegible]E[illegible]	31333.x1[illegible]613[illegible]	5.73	0.0002
ERROR	43	2[illegible].5647[illegible]	5[illegible].6A755319		
TOTAL	49	423[illegible].07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-145.58095706				
LIFE1	14.47644[illegible]	15.[illegible]13[illegible]	[illegible]41.665505252	0.09	0.3519
LIFE2	-16.73953243	8.31[illegible]1565	22173.[illegible]3552[illegible]	4.06	0.0551
LIFE3	2.63529406	2.[illegible]7256[illegible]	4692.19844030	0.86	0.3598
LIFE4	-2.01139982	[illegible].0708794[illegible]	5166.[illegible]5576[illegible]	0.95	0.3364
LIFE5	4.21[illegible]5541	2.9[illegible]1[illegible]	11220.2[illegible]5047	2.05	0.1592
HS	4.54[illegible]743	[illegible].0006[illegible]	11[illegible].[illegible]9e326050	20.62	0.0001

MODEL IS THE BEST 6 VARIABLE MODEL FOUND.

VARIABLE IE ENTERED R SQUARE = 0.[illegible]319095

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	7	2[illegible]31.[illegible]A[illegible]21A	27200.[illegible]46C	5.61	0.0001
ERROR	42	2[illegible]23.1[illegible]1336	5[illegible].31395270		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-169.[illegible]205175				
LIFE1	14.21[illegible]569	15.[illegible]0945[illegible]	4[illegible].00762[illegible]	0.90	0.3491
LIFE2	-14.[illegible]98750	[illegible].2139[illegible]	16[illegible].C9039634	3.09	0.0862
LIFE3	2.[illegible]61145	2.[illegible]11175	3952.[illegible]201967	0.76	0.3889
LIFE4	-2.[illegible]39576	5.[illegible]13[illegible]	5550.[illegible]73158	1.07	0.3017
LIFE5	3.[illegible]44[illegible]17	2.[illegible]3[illegible]	7[illegible].[illegible]1075[illegible]	1.41	0.2419
HS	3.97571654	1.02771387	77839.[illegible]5729550	14.97	0.[illegible]06
IE	4.58906481	2.58527934	16401.37877388	3.15	0.0831

MODEL IS THE BEST 7 VARIABLE MODEL FOUND.

VARIABLE AVAT ENTERED R SQUARE = 0.49236928

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	8	208233.01063206	26036.62632901	4.97	0.0002
ERROR	41	214[illegible].06492360	5237.36743716		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-203.[illegible]5[illegible]619				
LIFE1	14.[illegible]7[illegible]589	15.05[illegible]2[illegible]95	4771.35333725	0.91	0.3456
LIFE2	-14.[illegible]59[illegible]23	8.[illegible]3[illegible]893	15577.[illegible]7979081	2.97	0.0921
LIFE3	2.[illegible]063677	2.[illegible]55[illegible]35	4256.[illegible]8[illegible]216	0.81	0.3737
LIFE4	-2.[illegible]4[illegible]33	[illegible].[illegible]564[illegible]77	7034.[illegible]7[illegible]230	1.34	0.2532
LIFE5	3.[illegible]03[illegible]39	2.[illegible]22[illegible]915	7[illegible].[illegible]66[illegible]761	1.39	0.[illegible]48
AVAT	5.[illegible]9[illegible]367	6.[illegible]6[illegible]35	[illegible]891.[illegible]2[illegible]8[illegible]	0.74	0.3937
HS	3.[illegible]26652	1.[illegible]2[illegible]52	76[illegible].[illegible]7[illegible]201	14.[illegible]	0.[illegible]05
IE	4.[illegible]7[illegible]362	2.[illegible]7[illegible]06	[illegible]698.[illegible]1949257	[illegible]	0.[illegible]659

MODEL IS THE BEST 8 VARIABLE MODEL FOUND.

(table continues)

VARIABLE ADAT ENTERED R SQUARE = 0.44971462

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	9	211471.46666446	23496.827407383	4.44	0.0004
ERROR	40	211253.89188889	5281.3472222222		
TOTAL	49	422725.35855556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-180.64236303				
LIFE1	18.54806781	15.212031251	852.91398644	1.04	0.3139
LIFE2	-16.34372261	6.3844474115	1.2E+3	3.79	0.0511
LIFE3	2.17666244	2.4893313135	31.5	0.59	0.4476
LIFE4	-2.13501357	3.0657766217	674E+1	1.28	0.4666
LIFE5	3.34557176	2.9293571130	6900.9256767	1.31	0.3622
AVAT	1.69174341	0.4E656E+0	4073.4721979	0.77	0.3855
ADAT	-0.37ce6440	0.4993968454	3104.9566259	0.59	0.4478
MS	1.9714E549	1.0316E5317	77173.725193058	14.62	0.0005
IE	4.0618109595	2.0647915E5E	17635.698522546	3.37	0.0738

MODEL IS THE BEST 9 VARIABLE MODEL FOUND.

VARIABLE ADSI ENTERED R SQUARE = 0.50457541

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	10	213444.34484E15E	21344.834484E923	3.97	0.0009
ERROR	39	209377.51E9E1E31	5373.7b9919449		
TOTAL	49	423225.07855556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-185.21944573				
LIFE1	18.5E5E52733	15.331133316	5494.4e312730	1.02	0.3162
LIFE2	-16.5E1945931	6.4647E52E14	1777E.9563E158	3.31	0.0766
LIFE3	2.2E3130E394	2.9316E1E749	4022.7315E362	0.75	0.3922
LIFE4	-2.2E296A6316	3.1E4E93E746	6E15.0678B076	1.21	0.2775
LIFE5	3.4E2HE31E43	3.1E4E9E4E6E	5E+3.8E265E23	1.67	0.2175
AVAT	1.6491E57E85	0.8E8E489741	1493.0368E631	0.28	0.4674
ADAT	-0.3E8E7315E73	0.5E1E170716	9371.4E746E13	0.81	0.3726
AVSI	0.2E67195E6	0.4E7E4E1E60	2E4E.9E5E3E45	0.38	0.5408
MS	1.E3E7E3E144	1.2E76721E5	6E8E+1.1E75E67	12.77	0.0010
IE	4.7E91E7E514	2.1E7138E1E5	17E7E+1.4E38184	3.17	0.0663

MODEL IS THE BEST 10 VARIABLE MODEL FOUND.

VARIABLE AVSI ENTERED R SQUARE = 0.53e69976

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	11	215196.22E42667	19536.7e752773	3.5e	0.0016
ERROR	38	207947.8E7E5043	5470.e49e12502		
TOTAL	49	423225.07E55556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-163.131e7724				
LIFE1	16.4e9775E0	15.E0456462	5491.2164246e	1.00	0.3056
LIFE2	-16.E622E265	6.3770e1E67	1877E.47E5E963	3.43	0.0717
LIFE3	2.2E96E5E743	2.9444e1E47	4221.1e4099E2E	0.77	0.3852
LIFE4	-2.2437e53736	3.10b1E4E4E2	6919.1022E5263	1.27	0.2671
LIFE5	3.9395E4e6E4	3.1E4E9E0E67	6973.E52464E3	1.28	0.2696
AVAT	1.6E2E1E955C	0.5E4E6E7E61	4643.e6235387E	0.84	0.3590
ADAT	-0.7E11166	0.5E8E5E4e6A	5E30.e7e9E935	1.07	0.3E3A
AVSI	-0.5E43e93E59	0.6E4E05E1eA	169F.e1739125E	0.31	0.5807
ADSI	0.1221171E20	0.3171E1E19	3271.4966e641	0.60	0.4441
MS	1.7444E0574	1.1E2E2E611E	64E4E.1E2E9E27	11.75	0.0016
IE	4.6729995e5	2.7D1E53E5	16402E.0C358121	3.00	0.0914

MODEL IS THE BEST 11 VARIABLE MODEL FOUND.

VARIABLE HY ENTERED R SQUARE = 0.53959531

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	12	215571.59293106	17964.299410E2	3.20	0.0032
ERROR	37	207653.46716E45E	5606.85089174		
TOTAL	49	423225.07855556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-172.84556107				
LIFE1	17.95133214	17.08987245	6196.353431E2	1.10	0.3003
LIFE2	-16.63202908	6.9373A8E93	17203.83866851	3.43	0.0722
LIFE3	2.2243E1E0d	3.1E6085534	2947.3E033721	0.53	0.4729
LIFE4	-2.2E9663509	3.1E5E77E38	6337.e1641631	1.14	0.2924
LIFE5	3.7E7722E6	3.1E3377E75	7770.E1E03842	1.39	0.2466
AVAT	1.6A1321E2D	0.5E6A6E567	5429.e1463173	0.61	0.4352
ADAT	-0.E59E43C06	0.8E5E99E17	5998.e4255195	1.07	0.3077
AVSI	-0.4e8e8E5702	0.5E4115e6E	1346.2E0DC043	0.24	0.6270
ADSI	0.07E02198A	0.4E0369309	3166.e2618261	0.57	0.4570
MS	3.4E5E29E65	1.5E4161156	28d17.e7407017	5.16	0.0295
HY	0.7403111	1.1E33d5870	436.3702E639	0.08	0.7845
IE	4.7847E071	2.4732E5E4E	16797.81975744	3.00	0.0918

MODEL IS THE BEST 12 VARIABLE MODEL FOUND.

Note: In all steps of this procedure LIFE 1 - LIFE 5 were included as the first variables, i.e., forced into the first five positions in each model. All other independent variables were entered in a simultaneous stepwise fashion. LIFE 1 - LIFE 5 = life event factors; HS and HY = somatization factors; AVAT, ADAT, AVSI, and ADSI = social support factors; IE = locus of control.

This increase in correlation, and thus variance accounted for, is significant, F (7,37) = 3.57, $p < .01$, but somewhat misleading. Closer examination of Table 4 and the different models obtained as the various moderator variable subscales are entered in a nonhierarchical or simultaneous stepwise fashion reveals that the increase in the strength of the correlation is mainly attributable to the independent variable HS, the Hypochondriasis scale of the MMPI, one of the two measures of somatization. When HS is entered, the correlation jumps from .4219 for the LIFE 1 - LIFE 5 model to .6667, F (1,43) = 20.62, $p < .005$. However, when the variable IE (Rotter's I-E scale, the measure of locus of control) which accounts for the next highest proportion of variance of all the remaining independent variables, is entered the increase in the correlation coefficient to .6951 is not significant F (1,42) = 3.15, n.s. These results suggest that HS was the key factor in the increase in variance found for the twelve variable model and that the twelve variable model might not represent a significant increase in predictive power over the six variable model (LIFE 1 - LIFE 5 plus HS). Comparison of the variance accounted for by the six and twelve variable models reveals that the increase is in fact not a significant one, F (6,37) = 1.22, n.s. The same pattern is revealed in Table 5 which contains the analysis for the

Table 5

Regression Analysis of Life Event Factors, Locus of Control, Somatization Factors, and Social Support Factors on Physical Complaints, LIFE 1 - LIFE 5 Forced.

INCLUDED VARIABLES ENTERED R SQUARE = 0.24183496

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	25217.70049224	5043.54009845	3.66	0.0074
ERROR	44	60634.98013665	1377.38040538		
TOTAL	49	85822.68588889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	11.22473377				
LIFE1	-0.77026023	1.66792261	533.07001466	0.39	0.5371
LIFE2	1.11051180	1.07685224	118.91695579	0.09	0.7703
LIFE3	2.49446553	1.42921150	4195.65931320	3.05	0.0879
LIFE4	-0.58021814	1.01945515	442.47038870	0.32	0.5737
LIFE5	0.26662695	1.45479031	47.56787125	0.03	0.8534

VARIABLE MS ENTERED R SQUARE = 0.53107333

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	6	45715.90151506	7619.31691920	7.60	0.0001
ERROR	43	40106.78731363	932.67831101		
TOTAL	49	85822.68882869			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-76.77936353				
LIFE1	-1.35562969	6.43225424	42.64502650	0.04	0.8337
LIFE2	-0.66111117	1.07555047	1719.21133631	1.80	0.1869
LIFE3	2.43878253	1.19077457	4039.65331402	4.20	0.0467
LIFE4	-0.57179894	0.86623134	416.69713707	0.44	0.5126
LIFE5	1.58142396	1.33001774	1581.75506453	1.65	0.2052
MS	1.89325703	0.41244543	19532.21122323	20.49	0.0001

MODEL IS THE BEST 6 VARIABLE MODEL FOUND.

VARIABLE IE ENTERED R SQUARE = 0.55173663

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	7	47377.14674878	6768.15810697	7.38	0.0001
ERROR	42	38444.53921811	916.03719501		
TOTAL	49	85822.68888689			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-86.49221377				
LIFE1	-1.44631425	6.29713446	59.44090176	0.06	0.8174
LIFE2	-0.73716167	1.04467766	1577.18650048	1.72	0.2043
LIFE3	2.34139605	1.16670459	3775.10333269	4.12	0.0500
LIFE4	-0.60161483	0.84477148	460.77595654	0.50	0.4821
LIFE5	1.27470756	1.21793570	1631.44261620	1.78	0.3013
MS	1.68237285	0.41131759	13224.54966465	14.43	0.0006
IE	1.43736846	1.23452345	2629.23523512	2.87	0.0976

MODEL IS THE BEST 7 VARIABLE MODEL FOUND.

VARIABLE AVAT ENTERED R SQUARE = 0.55935282

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	8	48005.15221213	6000.64402652	6.51	0.0001
ERROR	41	37817.51667675	922.37845553		
TOTAL	49	85822.68888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-104.32033840				
LIFE1	-1.39AE3113	6.31334676	48.17739568	0.05	0.8259
LIFE2	-1.64850820	1.04532171	1016.34863697	1.11	0.2977
LIFE3	2.38476411	1.17150449	3878.22949513	4.16	0.0478
LIFE4	-0.72397315	0.86202469	647.55303435	0.70	0.4070
LIFE5	1.27018631	1.22315504	936.32239595	1.08	0.3048
AVAT	2.2833P7EH	2.70743349	656.34546336	0.71	0.4039
MS	1.64486785	0.41311367	13322.31657635	14.42	0.0005
IE	1.59414308	1.19403731	3009.32236863	3.26	0.0782

MODEL IS THE BEST 8 VARIABLE MODEL FOUND.

(table continues)

VARIABLE HY ENTERED R SQUARE = 0.56094665

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	9	48141.92366458	5349.10329611	5.68	0.0001
ERROR	40	37680.73923391	942.01848060		
TOTAL	49	85822.66288849			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-105.13707776				
LIFE1	-0.37065556	6.93163025	2.69228815	0.00	0.9576
LIFE2	-3.66710850	1.54267514	1123.45633330	1.19	0.2815
LIFE3	2.13901773	1.24905419	3074.09475421	3.26	0.0807
LIFE4	-0.66667834	0.97731476	577.47562756	0.61	0.4383
LIFE5	1.17545433	1.26987179	820.30823772	0.87	0.3564
AVAT	2.13651287	2.76316217	563.18543478	0.60	0.4439
HS	1.48335348	2.61400516	5477.36909512	5.81	0.0206
HY	2.64702306	0.94672165	176.37745284	0.19	0.6652
IE	2.64101076	1.12204683	3114.49456832	3.31	0.0765

MODEL IS THE BEST 9 VARIABLE MODEL FOUND.

VARIABLE ADAT ENTERED R SQUARE = 0.56104006

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	10	48153.38342136	4815.33834214	4.99	0.0001
ERROR	39	37668.28046950	965.87898635		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-103.91506206				
LIFE1	-0.26037531	7.08251673	1.33173195	0.00	0.9709
LIFE2	-3.80030154	1.59035207	1129.86956890	1.17	0.2859
LIFE3	2.21131517	1.26731091	2851.59177485	2.95	0.0937
LIFE4	-0.68287115	0.98624288	568.91910123	0.59	0.4476
LIFE5	1.16644533	1.27886858	804.10741590	0.83	0.3672
AVAT	2.13304109	2.78805411	564.48025213	0.58	0.4492
ADAT	-0.02317882	0.21662601	11.45875641	0.01	0.9138
HS	1.47706298	2.65211305	5431.56595083	5.63	0.0227
HY	2.74573139	2.95234978	194.76576453	0.20	0.6588
IE	2.71568713	1.11781461	3054.63151500	3.16	0.0812

MODEL IS THE BEST 10 VARIABLE MODEL FOUND.

VARIABLE ADSI ENTERED R SQUARE = 0.56117781

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	11	48161.77739382	4376.34339641	4.42	0.0003
ERROR	38	37660.88949507	991.07669344		
TOTAL	49	85822.66688889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-104.23312301				
LIFE1	-0.65512815	7.19461078	1.25561814	0.00	0.9718
LIFE2	-3.77377243	1.76647026	1122.71174438	1.11	0.2582
LIFE3	2.23255778	1.35590834	2619.37074816	2.64	0.0999
LIFE4	-0.67973374	1.01125642	563.74927049	0.57	0.4554
LIFE5	1.17669363	1.33605710	795.15192216	0.80	0.3761
AVAT	2.06672932	2.93778567	516.44225538	0.52	0.4748
ADAT	-0.02892254	0.22445341	16.96764458	0.02	0.8981
ADSI	0.14711104	0.59916674	6.24892614	0.01	0.9272
HS	1.46691317	2.99455646	5136.90413307	5.14	0.0277
HY	2.76463489	2.96714387	146.66601112	0.15	0.7026
IE	2.02962018	1.15426745	3064.85066728	3.09	0.0867

MODEL IS THE BEST 11 VARIABLE MODEL FOUND.

VARIABLE AVSI ENTERED R SQUARE = 0.56135583

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	12	48177.05533750	4014.75461146	3.95	0.0006
ERROR	37	37645.61355119	1017.44901490		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-101.84030895				
LIFE1	-0.26613889	7.26307153	1.35974677	0.00	0.9710
LIFE2	-4.31033691	1.92944803	1116.33438977	1.10	0.3017
LIFE3	2.24910063	1.34789271	2832.95973647	2.78	0.1036
LIFE4	-0.64966155	0.98167586	575.80477557	0.57	0.4566
LIFE5	1.21232865	1.35605674	809.22290223	0.80	0.3785
AVAT	2.20913926	3.09465672	516.49230406	0.51	0.4798
ADAT	-0.03874472	0.24161055	26.27411303	0.03	0.8752
AVSI	-0.17369230	1.23268431	15.27798798	0.02	0.9031
ADSI	0.22572039	1.74258400	17.07133816	0.02	0.8976
HS	1.46891180	0.64924810	5208.14089095	5.12	0.0296
HY	2.57696976	2.90956586	129.96188855	0.12	0.7280
IE	2.01934769	1.17264174	3017.19528484	2.97	0.0934

MODEL IS THE BEST 12 VARIABLE MODEL FOUND.

Note: In all steps of this procedure LIFE 1 - LIFE 5 were included as the first variables, i.e., forced into the first five positions in each model. All other independent variables were entered in a simultaneous stepwise fashion. LIFE 1 - LIFE 5 = life event factors; HS and HY = somatization factors; AVAT, ADAT, AVSI, and ADSI = social support factors; IE = locus of control.

dependent variable of physical complaints. Again a significant increase in the strength of correlation is obtained when all the moderator variable subscales are entered. In this case, the correlation increases from .5421 for the five variable model LIFE 1 - LIFE 5 to .7492 for the best twelve variable model, F (7,37) = 3.23, $p < .05$. However, once more the change occurs mainly when HS is entered to produce the best six variable model with an increase in correlation over the five variable model from .5421 to .7219, F (1,43) = 20.42, $p < .005$. Similarly, I-E accounts for the next greatest amount of variance, but produces a seven variable model with a correlation coefficient of .7428 which is not significantly higher than that of the six variable model, F (1,42) = 2.87, n.s. The increase in the correlation from .7219 for the six variable model to .7492 for the twelve variable model is not significant, F (6,37) = .56, n.s., suggesting that for physical as well as psychological complaints, HS is the key factor in the increased variance accounted for with the addition of all of the moderator variables.

Search for the "Best" Model

To determine whether more parsimonious, yet powerful models could be found, simultaneous stepwise regression analyses were generated for each dependent variable, without any independent variables being forced into the

initial position as was done to assess the experimental hypotheses. The results for the criterion variable of psychological complaints presented in Table 6 reveal that HS accounts for the greatest amount of variance of all the independent variables, $\underline{R}$ = .5875, $\underline{R}^2$ = .3452, $\underline{F}$ (1,48) = 25.30, $\underline{p} < .0001$. When the variable I-E is entered to derive the best two variable model, the increase in correlation to .6275 is not significant, $\underline{F}$ = (1,47) 3.77, n.s. However, in an exploratory analysis such as this, the F-test for extra sum of squares might not be the most useful criterion to decide when to stop adding predictor variables to determine the "best" model. In order to find the model most relevant for intervention in the stress-illness process, one would want to examine various selection criteria to find a model which accounts for a high degree of variance in symptomatology while being parsimonious, and thus cost-effective. Another criterion which can be used to select the "best" set of independent variables is the MSEp criterion. The MSEp criterion calls for an examination of the mean square error term (MSE) in order to select the best set of independent variables. The number of parameters in the regression equation is shown as a subscript of MSE; therefore MSEp indicates there are $\underline{p}$ parameters or $\underline{p}$-1 independent variables. For this criterion, one seeks either to find the set of independent variables which

Table 6

Regression Analysis of Life Event Factors, Locus of Control, Somatization Factors, and Social Support Factors on Psychological Complaints.

VARIABLE HS ENTERED R SQUARE = 0.34517167

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	1	146016.27273832	146016.27273832	25.30	0.0001
ERROR	48	277338.00276774	5771.01672432		
TOTAL	49	423325.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-105.62788230				
HS	4.29412960	0.85170451	146016.27273832	25.30	0.0001

MODEL IS THE BEST 1 VARIABLE MODEL FOUND.

VARIABLE IE ENTERED R SQUARE = 0.39377106

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	2	166675.03162264	83337.51581102	15.26	0.0001
ERROR	47	256650.04393292	5460.38391348		
TOTAL	49	423325.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-135.89965323				
HS	3.84129793	0.85901353	109106.66334380	20.00	0.0001
IE	5.03754700	2.59521370	20512.75683372	3.77	0.0583

MODEL IS THE BEST 2 VARIABLE MODEL FOUND.

VARIABLE LIFE3 ENTERED R SQUARE = 0.42566628

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	3	180287.5109+501	60022.50365533	11.36	0.0001
ERROR	46	243037.56952055	5283.68618675		
TOTAL	49	423325.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-138.51961861				
LIFE3	1.22566359	0.7+685420	13+32.+7934297	2.55	0.1168
HS	3.1A497204	0.9399+2585	63+03.962254737	11.93	0.0014
IE	5.30869096	2.35639552	22731.11028157	4.30	0.0437

MODEL IS THE BEST 3 VARIABLE MODEL FOUND.

VARIABLE LIFE2 ENTERED R SQUARE = 0.+5341740

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	4	156727.+6561075	+7676.76639269	9.23	0.0001
ERROR	45	232510.51393+A3	5162.622445+00		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-142.00277311				
LIFE2	-6.22567477	+.33521276	10679.5+456575	2.06	0.1580
LIFE3	1.56375287	1.79649135	+2315.662244027	1.94	0.0534
HS	3.61963471	0.97726515	70850.93544263	13.72	0.0006
IE	4.95200277	2.54212347	19590.19079855	3.79	0.0577

LIFE3 REPLACED BY LIFE1 R SQUARE = 0.+5165790

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	4	151002.6181704+	+7765.+5+5+260	9.27	0.0001
ERROR	45	232+22.95736516	5154.72127523		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-168.31727287				
LIFE1	19.+7530512	9.+625+4922	20671.+15C98891	+.01	0.0513
LIFE2	-10.19073395	6.19347693	14137.+76C02+9	2.74	0.1047
HS	3.71564202	0.9+2+5141	73718.61542762	14.31	0.0005
IE	+.39870715	2.53879852	19+53.15717282	3.88	0.0551

MODEL IS THE BEST + VARIABLE MODEL FOUND.

(table continues)

VARIABLE LIFE5 ENTERED R SQUARE = 0.46246721

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	195643.66757178	39128.73351436	7.57	0.0001
ERROR	44	227385.30754178	5167.75608812		
TOTAL	49	423029.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-175.69315133				
LIFE1	20.64444362	9.73136781	23032.64478165	4.46	0.0465
LIFE2	-15.72902358	8.16121514	17996.66235777	3.48	0.0687
LIFE3	4.65647448	3.95146619	4581.06960135	0.89	0.3516
LIFE5	3.49341032	1.32751146	78211.47363112	15.13	0.0003
IE	4.62507555	2.57277631	16720.82705881	3.23	0.0791

MODEL IS THE BEST 5 VARIABLE MODEL FOUND.

VARIABLE LIFE4 ENTERED R SQUARE = 0.47384772

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	6	200469.46662231	33478.24443372	6.46	0.0001
ERROR	43	222565.61093324	5176.17697752		
TOTAL	49	423035.07755556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-191.15539438				
LIFE1	23.72122330	10.06261059	27849.59025131	5.38	0.0259
LIFE2	-14.81325130	8.17395663	15992.79319179	3.09	0.0770
LIFE4	-1.91347330	2.00815822	4826.77755054	0.93	0.3407
LIFE5	3.15194705	1.33134051	69511.03356985	13.43	0.0705
MS	3.67744053	1.32452709	77776.21055529	15.03	0.0004
IE	4.68572065	2.57165918	17131.15319431	3.31	0.0755

MODEL IS THE BEST 6 VARIABLE MODEL FOUND.

VARIABLE ADAT ENTERED R SQUARE = 0.4932769*

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	7	208436.26201262	29805.46600180	5.61	0.0001
ERROR	42	214599.81358294	5204.44796150		
TOTAL	49	423035.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-153.27170448				
LIFE1	23.55739754	10.31194951	27161.12167644	5.22	0.0275
LIFE2	-14.50786161	8.23184621	17193.35270136	3.30	0.0763
LIFE4	-1.83966753	2.01412994	4615.14121604	0.89	0.3517
LIFE5	3.15446607	1.33224847	6190.96155730	14.19	0.0016
ADAT	-0.43436271	0.36348772	3946.79649031	0.77	0.3863
MS	4.01650583	1.32496648	73381.37426274	15.25	0.0003
IE	4.53076145	2.58811362	16305.74874109	3.08	0.0868

MODEL IS THE BEST 7 VARIABLE MODEL FOUND.

VARIABLE AVAT ENTERED R SQUARE = 0.49238666

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	8	208332.39217651	26036.53377237	4.97	0.0002
ERROR	41	214702.68339905	5237.37378967		
TOTAL	49	423035.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-186.13732257				
LIFE1	24.09377644	10.36040190	28220.71453034	5.39	0.0253
LIFE2	-14.70946733	8.23104868	16736.19370859	3.19	0.0813
LIFE4	-2.18464706	2.01816579	5958.86351610	1.14	0.2924
LIFE5	3.13337959	2.99152416	6107.83377134	1.17	0.2865
AVAT	5.53384642	6.45060352	3854.48816630	0.74	0.3959
ADAT	-0.43677188	0.49556793	4236.05858302	0.81	0.3737
MS	3.97666337	1.33337232	77555.96115507	14.81	0.0004
IE	4.91760674	2.63356625	19250.92456669	3.68	0.0690

ADAT REPLACED BY LIFE3 R SQUARE = 0.49238528

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	8	208331.91063206	26036.48632901	4.97	0.0002
ERROR	41	214703.16492350	5237.26743716		
TOTAL	49	423035.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-203.63575619				
LIFE1	14.37270589	15.05624195	4771.35333725	0.91	0.3454
LIFE2	-14.21546623	8.24304491	15577.17979081	2.97	0.0921
LIFE3	2.51061677	2.79115455	4236.31884216	0.81	0.3737
LIFE4	-2.36361439	2.05666177	7034.97286232	1.34	0.2532
LIFE5	3.43803434	2.91224919	7286.49613761	1.39	0.2456
AVAT	5.56096367	6.45162635	3891.12108988	0.74	0.3937
MS	3.73226652	1.03210502	76024.07711201	14.52	0.0005
IE	4.97087382	2.63074706	18698.21549257	3.57	0.0659

MODEL IS THE BEST 8 VARIABLE MODEL FOUND.

(table continues)

VARIABLE ADAT ENTERED R SQUARE = 0.44973862

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	9	211431.4668#####	23492.[illegible]	4.44	0.0004
ERROR	40	211633.[illegible]	5290.67771667		
TOTAL	49	423325.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-180.64236380				
LIFE1	15.54806761	15.21201051	5526.9133####	1.04	0.3129
LIFE2	-14.34575261	8.38654412	14822.10782755	3.00	0.0912
LIFE3	2.17665244	2.03325[illegible]	3109.21[illegible]	0.59	0.4478
LIFE4	-2.31501357	2.06796617	6745.[illegible]	1.26	0.2655
LIFE5	3.34553776	2.92931740	6950.92[illegible]	1.30	0.2602
AVAT	5.69174341	6.48656082	4073.47199837	0.77	0.3855
ADAT	-0.37866468	0.49596855	3108.95625604	0.59	0.4478
HS	3.57135549	1.03862597	17373.72189069	14.62	0.0005
IE	4.26108598	2.64793696	17835.99552546	3.37	0.0738

MODEL IS THE BEST 9 VARIABLE MODEL FOUND.

VARIABLE ADSI ENTERED R SQUARE = 0.50457541

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	10	213448.[illegible]	21344.[illegible]	3.97	0.0009
ERROR	39	209577.[illegible]	5373.76991444		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-185.[illegible]				
LIFE1	18.50250733	15.33122116	5494.44312730	1.02	0.3182
LIFE2	-13.[illegible]	8.[illegible]	17716.[illegible]	3.30	0.0766
LIFE3	2.[illegible]	2.[illegible]	4022.[illegible]	0.75	0.3922
LIFE4	-2.[illegible]	2.[illegible]	6519.[illegible]	1.21	0.2778
LIFE5	3.82860183	3.[illegible]	6445.[illegible]	1.19	0.2775
AVAT	4.[illegible]	6.66446741	2894.[illegible]	0.54	0.4674
ADAT	-0.46735475	0.51817016	4371.46746610	0.81	0.3726
ADSI	2.23621905	1.72204155	2046.[illegible]	0.38	0.5408
HS	3.41620144	1.07167213	68643.[illegible]	12.77	0.0010
IE	4.75915314	2.67386255	17023.94230158	3.17	0.0829

MODEL IS THE BEST 10 VARIABLE MODEL FOUND.

VARIABLE AVSI ENTERED R SQUARE = 0.50658976

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	11	214246.[illegible]	19476.[illegible]	3.58	0.0016
ERROR	38	208778.[illegible]	5470.49612502		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-163.12182728				
LIFE1	16.08971260	15.50454562	5891.21642696	1.08	0.3059
LIFE2	-13.[illegible]	8.[illegible]	18778.[illegible]	3.43	0.0717
LIFE3	2.[illegible]	2.[illegible]	4221.[illegible]	0.77	0.3852
LIFE4	-2.[illegible]	2.[illegible]	6939.[illegible]	1.27	0.2671
LIFE5	3.93954464	3.38830[illegible]	8903.09246400	1.63	0.2098
AVAT	6.66149590	7.[illegible]	4049.[illegible]	0.74	0.3950
ADAT	-0.57611165	0.[illegible]	5830.[illegible]	1.07	0.3664
AVSI	-2.[illegible]	5.[illegible]	1698.17391212	0.31	0.5807
ADSI	3.12217128	4.[illegible]	3271.[illegible]	0.60	0.4441
HS	3.76420074	1.[illegible]	64285.[illegible]	11.75	0.0015
IE	4.67895045	2.70164365	16436.[illegible]	3.00	0.0914

MODEL IS THE BEST 11 VARIABLE MODEL FOUND.

VARIABLE HY ENTERED R SQUARE = 0.50959531

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	12	215571.[illegible]	17964.[illegible]	3.20	0.0032
ERROR	37	207453.[illegible]	5606.[illegible]		
TOTAL	49	423025.07555556			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-172.[illegible]				
LIFE1	17.[illegible]	17.[illegible]	6166.[illegible]	1.10	0.3003
LIFE2	-14.[illegible]	8.[illegible]	19233.[illegible]	3.43	0.0722
LIFE3	2.[illegible]	2.[illegible]	2947.[illegible]	0.53	0.4729
LIFE4	-2.[illegible]	2.[illegible]	6337.[illegible]	1.14	0.2924
LIFE5	3.[illegible]	3.[illegible]	7770.[illegible]	1.39	0.2466
AVAT	5.[illegible]	7.[illegible]	3649.[illegible]	0.61	0.4392
ADAT	-0.[illegible]	0.[illegible]	5398.[illegible]	1.07	0.3077
AVSI	-1.[illegible]	5.[illegible]	1346.[illegible]	0.24	0.6270
ADSI	3.[illegible]	4.[illegible]	1168.[illegible]	0.57	0.4570
HS	3.[illegible]	1.[illegible]	28811.[illegible]	5.14	0.0293
HY	4.[illegible]	17.[illegible]	455.[illegible]	0.08	0.7845
IE	4.76476601	2.[illegible]	16797.[illegible]	3.00	0.0918

MODEL IS THE BEST 12 VARIABLE MODEL FOUND.

Note: All independent variables were entered in a simultaneous stepwise fashion. LIFE 1 - LIFE 5 = life event factors; HS and HY = somatization factors; AVAT, ADAT, AVSI, and ADSI = social support factors; IE = locus of control.

minimizes MSEp or the set for which MSEp is "so close to the minimum that adding more independent variables is not worthwhile" (Neter & Wasserman, 1974, p. 379). The MSEp values for the best one variable through the best twelve variable model are plotted in Figure 4. The best four variable model, which includes HS, IE, LIFE 1, and LIFE 2, minimizes MSEp. It can be seen that there are no further reductions in MSEp with the addition of more independent variables. On the contrary, it increases since each successive reduction in SSEp was so small that it was not sufficient to offset the loss of an additional degree of freedom. Thus, this criterion would suggest inclusion of HS, IE, LIFE 1, and LIFE 2 as the "best" set of independent variables. This model does provide a significant increase in the correlation coefficient from that obtained with the single variable HS of .5875 to .6721, F (2,34) = 2.91, $p < .05$ whereas the full twelve variable model produces further increase only to .7139 which is not a significantly different, F (8.37) = 0.55, n.s. from the variance of the four variable model. The amount of variance accounted for by each of the best one through twelve variable models is visually displayed in Figure 5. A fairly sharp increase in variance is obtained between models with from one to four variables; thereafter, the increase in variance is more gradual with

Figure 4. Mean square error as a function of the number of independent varibles in regression models for the dependent variable of psychological complaints.

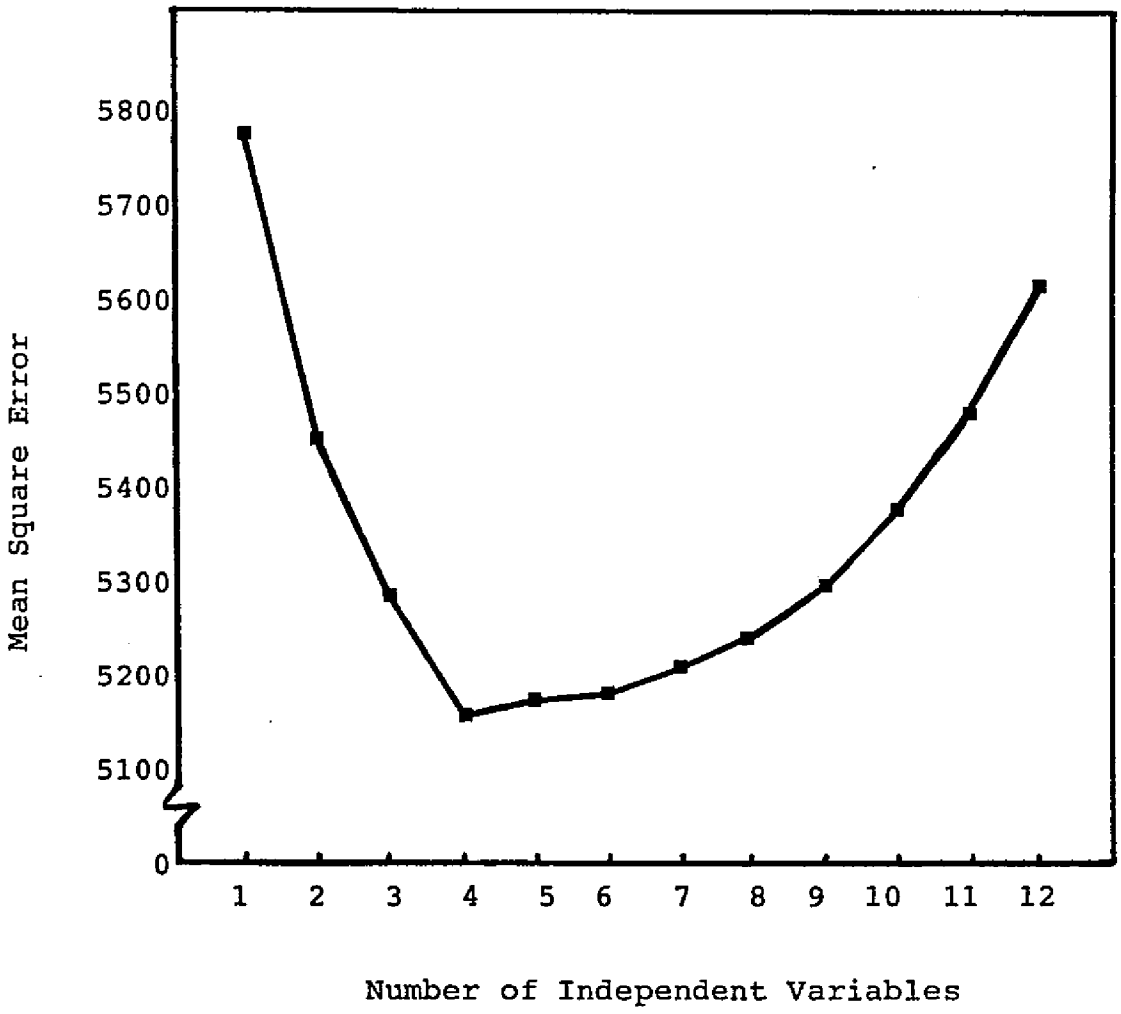
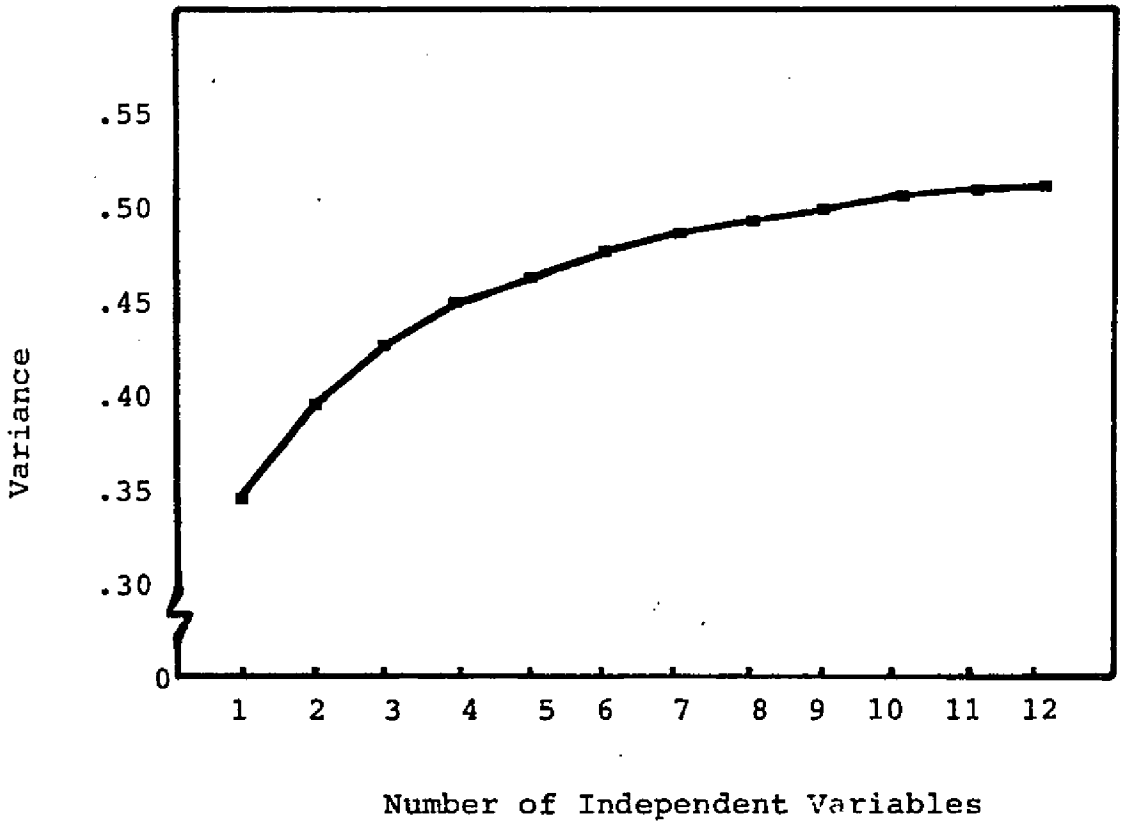


Figure 5. Variance (R^2) as a function of the numbers of independent variables in regression models for the dependent variable of psychological complaints.



the addition of successive variables. Thus, the four variable model (HS, IE, LIFE 1, LIFE 2) seems to account for a high degree of variance and is also parsimonious.

The results for the criterion variable of physical complaints are quite similar to those for psychological complaints. As seen in Table 7, HS again accounts for the greatest amount of variance of all the predictor variables with a correlation coefficient of .6264, $\underline{R}^2$ = .3923, $\underline{F}$ (1,48) = 30.99, $\underline{p} < .0001$. For physical complaints, the independent variable LIFE 3, the measure of change associated with life event occurrence, accounts for the next largest amount of variance with a significant increase in correlation to .6878, $\underline{F}$ (1,47) = 7.20, $\underline{p} < .05$. The addition of the variable IE increases the correlation coefficient of the regression equation to .7178 but this is not a significant change, $\underline{F}$ (1,46) = 4.01, n.s. In order to determine the "best" model for prediction of physical complaints, the MSEp values and the variances ($\underline{R}^2$) for the best one through twelve variable models are shown in Figures 6 and 7 respectively. The model that minimizes MSEp is the four variable model, which includes the predictor variables HS, LIFE 3, I-E, and LIFE 2. It can be seen from Figure 7 that the increase in variance accounted for begins to become much more gradual with increases in the size of the model beyond the four variable model. This four

Table 7

Regression Analysis of Life Event Factors, Locus of Control, Somatization Factors, and Social Support Factors on Physical Complaints.

VARIABLE MS ENTERED R SQUARE = 0.39232647

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	1	33670.5144[illegible]	33670.5144[illegible]	30.99	0.0001
ERROR	48	52152.1604[illegible]	1086.5333[illegible]		
TOTAL	49	85822.6868[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-70.9[illegible]				
MS	2.0[illegible]	0.3695[illegible]	33670.5[illegible]	30.99	0.0001

MODEL IS THE BEST 1 VARIABLE MODEL FOUND.

VARIABLE LIFE3 ENTERED R SQUARE = 0.47302849

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	2	40596.6317[illegible]	20298.3206[illegible]	21.09	0.0001
ERROR	47	45226.1573[illegible]	962.2[illegible]		
TOTAL	49	85822.6868[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-71.65692654				
LIFE3	0.87[illegible]	0.3166[illegible]	6926.05695139	7.20	0.0100
MS	1.60509729	0.3364[illegible]	16598.4[illegible]	17.25	0.0001

MODEL IS THE BEST 2 VARIABLE MODEL FOUND.

VARIABLE IE ENTERED R SQUARE = 0.51526304

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	3	44221.2[illegible]	14740.4[illegible]	16.30	0.0001
ERROR	46	41601.4[illegible]	904.3[illegible]		
TOTAL	49	85822.6[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-84.[illegible]				
LIFE3	0.91[illegible]	0.3[illegible]	7574.[illegible]	8.38	0.0058
MS	1.39730625	0.3[illegible]	11675.06693223	12.91	0.0008
IE	2.11[illegible]	1.05[illegible]	3624.[illegible]	4.01	0.0512

MODEL IS THE BEST 3 VARIABLE MODEL FOUND.

VARIABLE LIFE2 ENTERED R SQUARE = 0.53578115

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	4	45983.1[illegible]	11495.[illegible]	12.98	0.0001
ERROR	45	39839.[illegible]	885.3[illegible]		
TOTAL	49	85822.6[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-85.[illegible]				
LIFE2	-2.53[illegible]	1.79[illegible]	1760.[illegible]	1.99	0.1663
LIFE3	1.86[illegible]	0.7[illegible]	5590.[illegible]	6.32	0.0156
MS	1.57[illegible]	0.4[illegible]	13374.[illegible]	15.15	0.0003
IE	1.97476343	1.05273122	3115.35275741	3.52	0.0672

MODEL IS THE BEST 4 VARIABLE MODEL FOUND.

VARIABLE LIFE5 ENTERED R SQUARE = 0.54504938

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	5	46777.5[illegible]	9355.[illegible]	10.54	0.0001
ERROR	44	39044.[illegible]	887.[illegible]		
TOTAL	49	85822.[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-88.22070179				
LIFE2	-[illegible]	2.[illegible]	2363.[illegible]	2.66	0.1096
LIFE3	1.97[illegible]	0.7[illegible]	6115.[illegible]	6.89	0.0119
LIFE5	1.10922381	1.17[illegible]	798.[illegible]	0.90	0.3485
MS	1.69[illegible]	0.42[illegible]	14167.[illegible]	15.97	0.0002
IE	1.81653874	1.06[illegible]	2571.[illegible]	2.90	0.0958

MODEL IS THE BEST 5 VARIABLE MODEL FOUND.

(table continues)

VARIABLE LIFE4 ENTERED R SQUARE = 0.55113231

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	6	47249.6455447[illegible]	7883.2743078[illegible]	8.80	0.0001
ERROR	43	38[illegible].02[illegible]187	895.89[illegible]5E79		
TOTAL	49	85[illegible].66[illegible]889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-88.63324196				
LIFE2	-4.1775[illegible]416	[illegible].[illegible]E[illegible]669	1930.16[illegible]1[illegible]375	2.15	0.1450
LIFE3	2.[illegible]56[illegible]4[illegible]03	0.7[illegible]3[illegible]5[illegible]5E6	6635.[illegible]5524[illegible]313	7.41	0.0093
LIFE4	-0.6[illegible]3[illegible]5[illegible]557	0.[illegible]5[illegible]6[illegible]3[illegible]1[illegible]	[illegible].[illegible]0[illegible]2[illegible]4[illegible]71	0.58	0.4494
LIFE5	1.25[illegible]3[illegible]336	1.[illegible]3[illegible]1[illegible].[illegible]76	1038.[illegible]1[illegible]4[illegible]353	1.16	0.2877
MS	1.6741[illegible]237	0.[illegible]3[illegible]4[illegible]747	13937.[illegible]30[illegible]5[illegible]724	15.[illegible]	0.0003
IE	1.83[illegible]05725	1.[illegible]3[illegible]7971	2622.[illegible]3[illegible]5758[illegible]35	2.93	0.0945

MODEL IS THE BEST 6 VARIABLE MODEL FOUND.

VARIABLE AVAT ENTERED R SQUARE = 0.[illegible]8[illegible]2[illegible]2

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	7	47[illegible]53.[illegible]7[illegible]1[illegible]46	6851.[illegible]2[illegible]497[illegible]78	7.60	0.0001
ERROR	42	37[illegible]1.[illegible]9[illegible]40[illegible]743	901.[illegible]4927[illegible]601		
TOTAL	49	85[illegible]22.[illegible]6[illegible]3[illegible]d[illegible]3[illegible]869			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-102.[illegible]1[illegible]72213				
LIFE2	-4.[illegible]663[illegible]311	[illegible].[illegible]457[illegible]5[illegible]6[illegible]	1[illegible]7.5[illegible]299572	2.03	0.1619
LIFE3	2.[illegible]015[illegible]2[illegible]8	0.[illegible]7[illegible]6[illegible]3[illegible]627	6[illegible].[illegible]4[illegible]507[illegible]83	7.64	[illegible].0085
LIFE4	-0.[illegible]752[illegible]026	[illegible].[illegible]4[illegible]2[illegible]7[illegible]317	715.[illegible]4[illegible]126762	0.80	0.3768
LIFE5	1.[illegible]2[illegible]4[illegible]321[illegible]7	1.[illegible]2[illegible]3[illegible]4[illegible]091	1029.[illegible]5[illegible]290[illegible]55	1.14	0.2913
AVAT	[illegible].[illegible]3[illegible]2[illegible]61[illegible]1	[illegible].[illegible]6[illegible]7[illegible]4[illegible]6[illegible]E[illegible]	660.[illegible]3[illegible]69[illegible]4[illegible]	0.73	0.3969
MS	1.[illegible]05[illegible]7[illegible]521	[illegible].[illegible]2[illegible]54[illegible]1[illegible]E[illegible]	11[illegible]6[illegible].[illegible]7[illegible]711668	13.[illegible]	0.0003
IE	1.[illegible]99[illegible]2[illegible]087	1.[illegible]3[illegible]1[illegible]43[illegible]4[illegible]	3[illegible]03.[illegible]7[illegible]922230	3.33	[illegible].0751

MODEL IS THE BEST 7 VARIABLE MODEL FOUND.

VARIABLE MY ENTERED R SQUARE = 0.[illegible]6[illegible]1517

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	8	46[illegible]39.[illegible]21[illegible]92[illegible]683	6017.[illegible]43[illegible]61335	6.55	0.0001
ERROR	41	37[illegible]83.[illegible]31[illegible]3[illegible]736	919.[illegible]10809712		
TOTAL	49	85[illegible]22.[illegible]6[illegible]8[illegible]8[illegible][illegible]8[illegible]			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-105.[illegible]65[illegible]3[illegible]627				
LIFE2	-3.[illegible]97[illegible]61[illegible]73	2.[illegible]33[illegible]66[illegible]716	1733.[illegible]17051055	1.89	0.1772
LIFE3	2.[illegible]1[illegible]791332	[illegible].[illegible]8[illegible]2[illegible]1[illegible]33[illegible]	6734.[illegible]37679268	7.38	0.0096
LIFE4	+0.[illegible]691524[illegible]4	[illegible].[illegible]96[illegible]1[illegible]4[illegible]72[illegible]	591.[illegible]31[illegible]20873	0.[illegible]4	[illegible].[illegible]471
LIFE5	1.[illegible]17[illegible]16[illegible]81	1.[illegible]24[illegible]4[illegible]1[illegible]1[illegible]	810.[illegible]99[illegible]06491	0.[illegible]9	[illegible].[illegible]508
AVAT	2.[illegible]13[illegible]1[illegible]980	2.[illegible]72[illegible]82[illegible]55[illegible]	560.[illegible]72351172	0.61	0.4392
MS	1.[illegible]4[illegible]24[illegible]78[illegible]	0.[illegible]39[illegible]5[illegible]4[illegible]72	5651.[illegible]17[illegible]82[illegible]19	6.15	0.0174
MY	2.[illegible]7[illegible]15[illegible]063	6.[illegible]22[illegible]35[illegible]69	179.[illegible]66[illegible]79037	0.20	[illegible].[illegible]6611
IE	2.[illegible]4[illegible]15[illegible]77	1.[illegible]10[illegible]64[illegible]62	3124.[illegible]99267558	3.40	0.0724

MODEL IS THE BEST 8 VARIABLE MODEL FOUND.

VARIABLE ADAT ENTERED R SQUARE = 0.[illegible]51[illegible]64[illegible]9

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	9	48[illegible]3[illegible].[illegible]0[illegible]46[illegible]9[illegible]47	[illegible]350.[illegible]31[illegible]5[illegible]43[illegible]	5.68	0.0001
ERROR	40	37[illegible]7[illegible].[illegible]5[illegible]2[illegible]15[illegible]4[illegible]	941.[illegible]76[illegible]55[illegible]97		
TOTAL	49	85[illegible]22.[illegible]65[illegible]E[illegible]6[illegible]869			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-104.[illegible]36[illegible][illegible]2[illegible]353				
LIFE2	-3.[illegible]57[illegible]147[illegible]23	2.[illegible]3[illegible]1[illegible]56[illegible]668	1714.[illegible]8632[illegible]654	1.82	0.1848
LIFE3	2.[illegible]17[illegible]6[illegible]3[illegible]18	[illegible].[illegible]4[illegible]2[illegible]95[illegible]165	6[illegible]14.[illegible]0[illegible]257095	7.02	[illegible].[illegible]115
LIFE4	-0.[illegible]68[illegible]5[illegible]2[illegible]42	[illegible].[illegible]4[illegible]7[illegible]4[illegible]18[illegible]	579.[illegible]2[illegible]3671[illegible]41	0.61	[illegible].[illegible]4376
LIFE5	1.[illegible]16[illegible]5[illegible]5967	1.[illegible]1[illegible]61[illegible]4[illegible]366	[illegible]402.[illegible]4[illegible]588223	0.85	[illegible].[illegible]3613
AVAT	2.[illegible]13[illegible]77[illegible]14	2.[illegible]7[illegible]60[illegible]51[illegible]55	563.[illegible]20[illegible]199[illegible]24	0.60	[illegible].[illegible]4439
ADAT	-0.[illegible]32[illegible]2[illegible]9[illegible]04	[illegible].[illegible]2[illegible]7[illegible]9[illegible]0[illegible]69	12.[illegible]4[illegible]9[illegible]732[illegible]60	0.01	0.9076
MS	1.[illegible]47[illegible]2[illegible]40[illegible]5[illegible]	0.[illegible]3[illegible]2[illegible]6[illegible]03[illegible]4[illegible]	5633.[illegible]7[illegible]26[illegible]41[illegible]18	5.[illegible]9	0.0190
MY	2.[illegible]4[illegible]4[illegible]93[illegible]03	6.[illegible]41[illegible]7[illegible]8[illegible]107	145.[illegible]75[illegible]64[illegible]089	0.[illegible]0	[illegible].[illegible]6594
IE	2.[illegible]03[illegible]732[illegible]91	1.[illegible]12[illegible]73[illegible]257	3[illegible]1[illegible].[illegible]05[illegible]79[illegible]636	3.29	[illegible].[illegible]771

MODEL IS THE BEST 9 VARIABLE MODEL FOUND.

(table continues)

VARIABLE ADSI ENTERED R SQUARE = 0.56116318

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	13	48180.52177134	4816.05217313	4.99	0.0001
ERROR	39	37662.14716754	965.69680096		
TOTAL	49	85822.66893889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-134.67147746	3.11517171	1615.13754009	1.66	0.2022
LIFE2	-4.34638619	0.46130308	6257.53497307	6.48	0.0150
LIFE3	2.19775475	0.49776264	573.71970079	0.59	0.4455
LIFE4	-0.08272669	0.49776264	794.07556133	0.82	0.3731
LIFE5	1.19561117	1.31649741	515.15681392	0.53	0.4695
AVAT	2.30239242	2.48099545	18.13663463	0.02	0.8918
ADAT	-0.33006345	0.21340735	8.49782191	0.01	0.9260
ADSI	0.19752508	1.57845934	551.43203094	0.57	0.4551
HS	1.46221368	0.61123734	197.79168190	0.19	0.6617
HY	2.86776311	0.50143529	3071.06990055	3.18	0.0823
IE	2.23101680	1.13830867			

MODEL IS THE BEST 10 VARIABLE MODEL FOUND.

VARIABLE AVSI ENTERED R SQUARE = 0.56131938

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	11	48175.03554013	4379.63869007	4.42	0.0003
ERROR	38	37646.63334816	990.69298336		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-102.33477731				
LIFE1	-4.29465317	3.17548146	1660.27276106	1.68	0.2060
LIFE3	2.11223217	0.48615566	6247.13581484	6.31	0.0166
LIFE4	-0.67274156	0.49301597	5015.92787245	5.06	0.0300
LIFE5	1.21214432	1.31361459	809.12213841	0.82	0.3721
AVAT	2.06420016	2.45717366	517.15203547	0.52	0.4744
ADAT	-0.30596698	0.21339106	28.43781046	0.03	0.8667
AVSI	-0.67726979	2.20203954	15.17338938	0.02	0.9022
ADSI	0.22526115	1.71152913	17.09599432	0.02	0.8963
HS	1.46411946	0.62746461	5394.12529279	5.44	0.0250
HY	2.77409243	0.71546591	160.62442842	0.16	0.6894
IE	2.02062432	1.15661402	3023.76390113	3.05	0.0887

MODEL IS THE BEST 11 VARIABLE MODEL FOUND.

VARIABLE LIFE1 ENTERED R SQUARE = 0.56135583

	DF	SUM OF SQUARES	MEAN SQUARE	F	PROB>F
REGRESSION	12	48177.05531750	4014.75461146	3.95	0.0006
ERROR	37	37645.61355139	1017.44901490		
TOTAL	49	85822.66888889			

	B VALUE	STD ERROR	TYPE II SS	F	PROB>F
INTERCEPT	-101.64308695				
LIFE1	-0.26613989	7.28007153	1.35974677	0.00	0.9710
LIFE2	-4.31.23691	3.48264803	1116.13838977	1.10	0.3017
LIFE3	2.24910263	1.57796071	2822.98973647	2.78	0.1036
LIFE4	-0.68966155	2.91675860	575.80477557	0.57	0.4566
LIFE5	1.21332065	1.36050674	809.22290223	0.80	0.3783
AVAT	2.20513926	3.57465642	518.48230406	0.51	0.4798
ADAT	-0.01674472	0.24113425	26.27411303	0.03	0.8732
AVSI	-0.27399233	2.23268325	19.27798798	0.02	0.9031
ADSI	0.22572058	1.74256426	17.07130816	0.02	0.8976
HS	1.48891180	0.64924816	5208.14389095	5.12	0.0296
HY	2.59696976	7.40956586	124.98586899	0.12	0.7280
IE	2.01934769	1.17264274	2017.19528484	2.97	0.0934

MODEL IS THE BEST 12 VARIABLE MODEL FOUND.

Note: All independent variables were entered in a simultaneous stepwise fashion. LIFE 1 - LIFE 5 = life event factors; HS and HY - somatization factors; AVAT, ADAT, AVSI, and ADSI = social support factors; IE = locus of control.

Figure 6.- Mean square error as a function of the number of independent varibles in regression models for the dependent variable of physical complaints.

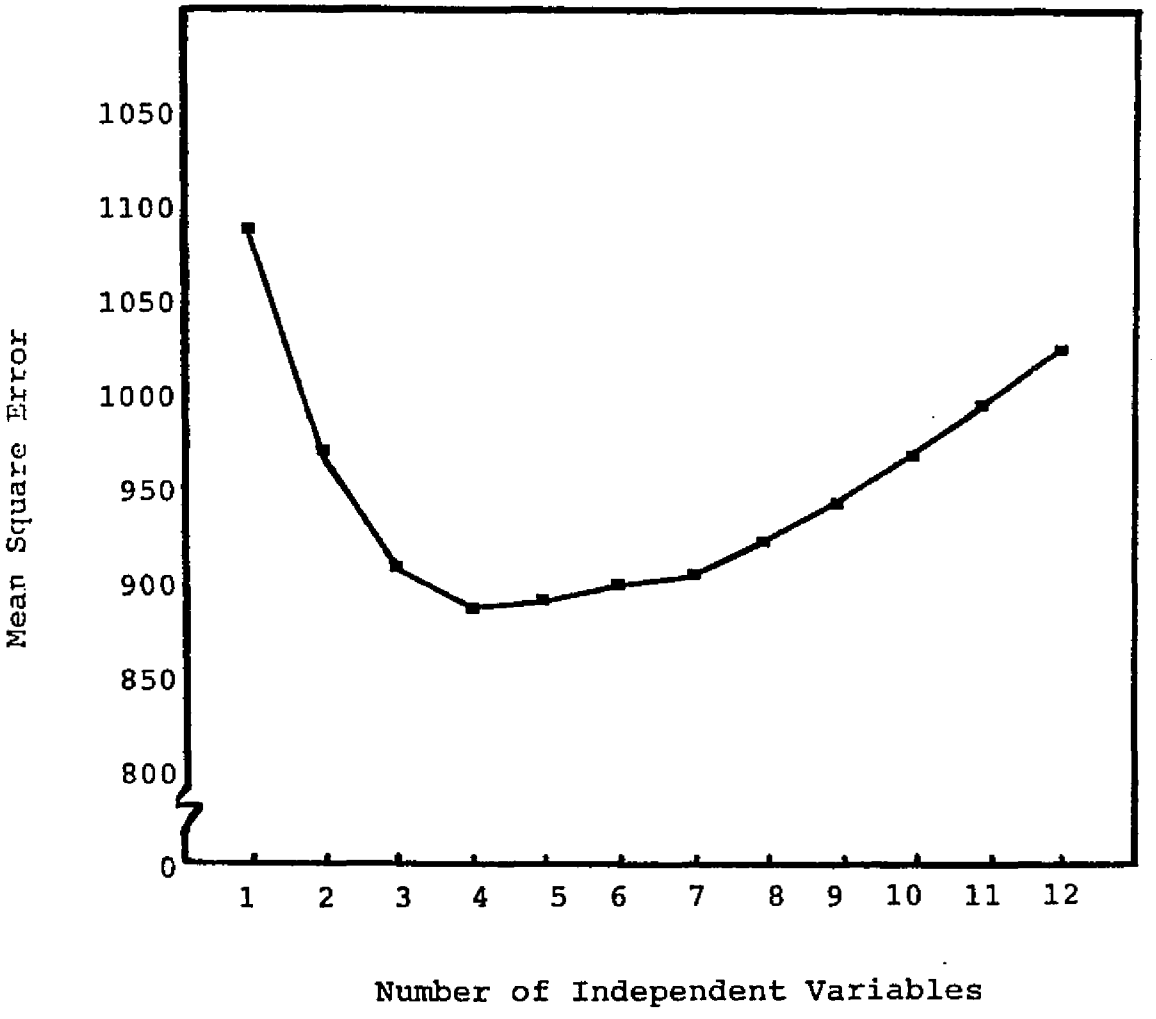
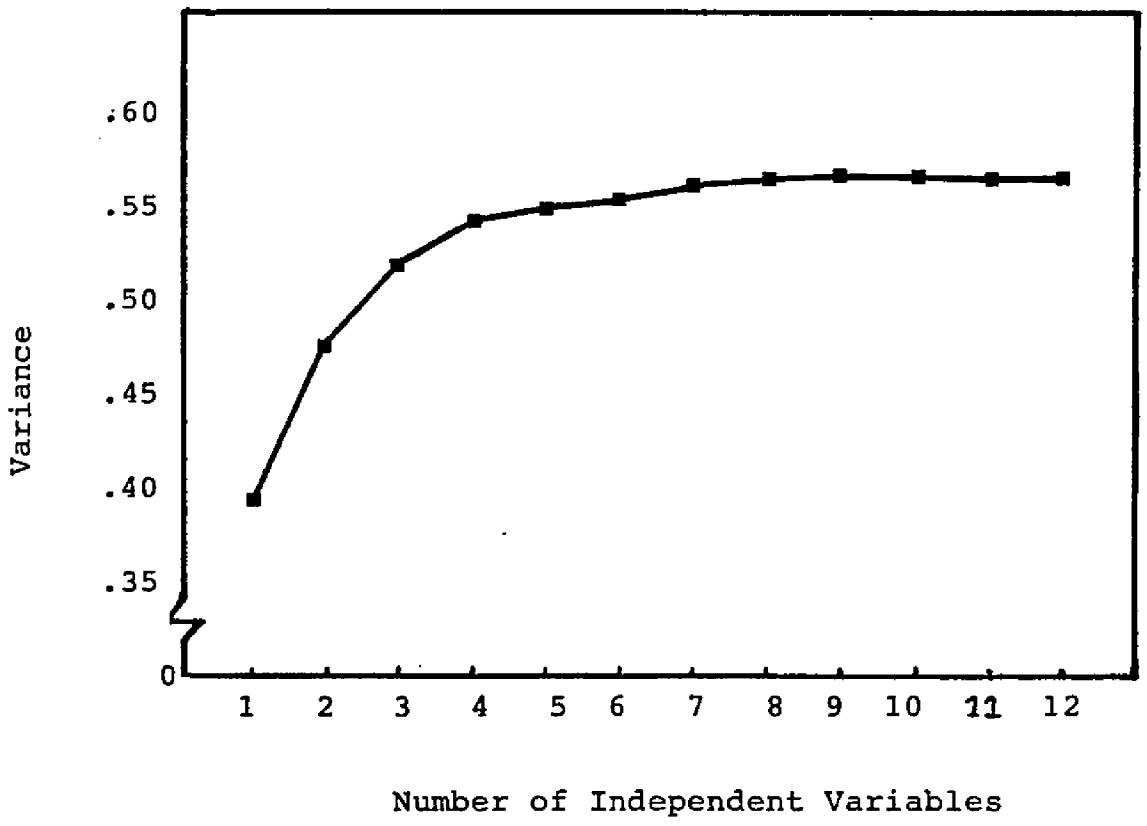


Figure 7. Variance (R^2) as a function of the number of independent variables in regression models for the dependent variable of physical complaints.



variable model provides a significant increase in the correlation from .6264 of the one variable (HS) model to .7320, F (3,45) = 4.64, $p < .05$. The full twelve variable model has a correlation coefficient of .7492 which is not significantly greater than that of the four variable model, F (8.37) = 0.27, n.s. These results suggest that the four variable model is the "best" model in terms of being both most parsimonious and powerful.

Comparison of the full twelve variable models for the tests of Hypothesis II and for the exploratory analyses reveals that the full models of both analyses account for the same amount of variance for each criterion variable. Thus, the total amount of variance accounted for was the same regardless of whether LIFE 1 - LIFE 5 was forced in first or not. However, the exploratory analyses were important for two reasons: First, they further clarified the relative significance of the predictor variable HS which had been suggested by the test of Hypothesis II; and second, they provided a more parsimonious yet powerful set of predictor variables for each criterion variable.

Descriptive Statistics for Test Scores

The descriptive statistics for the test scores in this study are presented in Table 8 and the intercorrelations between independent and dependent

Table 8

Descriptive Statistics for Criterion and Predictor Variables

Variable	Mean	SD	Range
Complaints			
Psychological	150.31	92.91	26 - 356
Physical	51.95	41.85	6 - 177
Life Events (LES)[a]			
Occurrence	9.90	4.00	3 - 22
Desirability	14.78	6.47	5 - 35
Change	31.37	15.05	8 - 83
Anticipation	22.75	11.88	5 - 54
Control	21.75	10.51	5 - 54
Social Supports (ISSI)			
Availability of Attachment	5.69	1.63	1 - 8
Adequacy of Attachment	61.54	21.98	17 - 100
Availability of Social Integration	8.77	2.44	2 - 13
Adequacy of Social Integration	9.90	3.33	2 - 18
Somatization (MMPI Scales)			
Hypochondriasis	59.74	12.74	33 - 95
Hysteria	52.80	10.70	30 - 90
Locus of Control			
I-E Scale	11.26	4.22	3 - 22

[a]Experimental version.

Table 9

Correlations of Life Event Factors, Social Support Factors, Somitization Factors, and Locus of Control with Psychological and Physical Complaints

	Complaints	
Predictor Variable	Psychological	Physical
Life Events (LES)[a]		
Occurrence	.39**	.49***
Desirability	.36*	.48***
Change	.40**	.53****
Anticipation	.30*	.42**
Control	.33*	.44**
Social Supports (ISSI)		
Availability of Attachment	.04	.05
Adequacy of Attachment	-.14	-.10
Availability of Social Integration	-.04	-.05
Adequacy of Social Integration	.11	.05
Somatization (MMPI Scales)		
Hypochondriasis	.59****	.63****
Hysteria	.44**	.51***
Locus of Control		
I-E Scale	.37**	.35*

[a]Experimental version.

*p .05. **p .01 ***p .001 ****p .001

measures in Table 9. Although no other studies using this experimental version of the LES have been published, the mean number of total events (LIFE, $\underline{X}$ = 9.90, $\underline{SD}$ = 4.0) is comparable to that found with the original version of the LES (I. G. Sarason, personal communication, December 4, 1984) and with other measures of life events (e.g., Masuda & Holmes, 1978; Rabking & Streuning, 1978). The mean number of negative events ($\bar{X}_{LIFE\ 2}$ - $\bar{X}_{LIFE\ 1}$ = 4.88) is also comparable to that found in other studies (e.g., Hurst, Jenkins, & Rose, 1978; Sandler & Lakey, 1982). The means found in this study for the ISSI subscales (AVAT $\bar{X}$ = 5.69, ADAT $\bar{X}$ = 61.54, AVSI $\bar{X}$ = 8.77, ADSI $\bar{X}$ = 9.90) are all within the average range found for the normative population (Henderson, Duncan-Jones, Byrne, & Scott, 1980) albeit at the lower end of the normal distribution. These scores have been found to increase with age (Henderson, et al., 1981). The mean T-Scores for the HS scale, $\bar{X}$ = 59.74, and the HY scale, $\bar{X}$ = 52.80, of the MMPI are within the average range for the normative population (Dahlstrom et al., 1972). Cooley and Keesey (1981, p. 714) reported "T scores of about 60 on the 1 [HS] scale and about 65 on the 3 [HY] scale" in their study of life events and illness in college students. According to Rotter (1975, p. 62) the mean I-E scale score for college students is "somewhere between 10 and 12, depending upon the sample." The IE mean of 11.26 for this study suggests this is a

typical college sample in terms of locus of control beliefs.

The interrelationships among all of the indepedent and the dependent measures are presented in Table 9. It should be noted that the two criterion measures, physical and psychological complaints, were highly correlated with each other, $r^2 = .89$, $p < .0001$. All the life event subscales were significantly correlated with both dependent measures as well as being highly correlated with each other. Correlations among the LES subscales ranged from .84 to .97. All LES subscales were significantly correlated with HS, $r = .40$, $p < .01$ to .50, $p < .001$, and with HY, $r = .30$, $p < .05$ to .41, $p < .01$. None of the LES subscales were correlated with locus of control (IE) nor any of the social support measures. The two measures of quantity of social support (AVAT = availability of attachment, AVSI = availability of social integration) were significantly correlated with each other, $r = .33$, $p < .005$, and the two measures of quality of social support (ADAT = adequacy of attachment, ADSI = adequacy of social integration) were also significantly correlated with each other, $r = .31$, $p < .005$. However, there were no other significant correlations of any of these social support measures with any other independent or dependent measure. Both of the somatization measures (HY and HS) were strongly and significantly correlated

with the dependent measures (see Table 9). It is not surprising that HS and HY are also highly correlated with each other, $r = .74$, $p < .0001$, since a majority of the HS items (20/33) overlap with HY and are scored in the same direction (Dahlstrom et al., 1972). As noted earlier, both HS and HY correlate significantly with all LES subscales. The correlations of HS and HY with the social support subscales and with locus of control were not significant. As seen in Table 9, locus of control (I-E Scale) was significantly correlated with both dependent variables. IE was not significantly correlated with any of the other predictor variables.

DISCUSSION

The study of the factors involved in the predisposition to, and precipitation of, illness is complicated by the fact that human functioning involves overt behavior, physiological processes, and symbolic activity in the context of a physical and social environment, all of which are interactive on an ongoing basis. Realistically, researchers can and do study only a portion of this process at one time. One area which has received increasing attention has been the role of psychosocial stress as a precursor to disease, hypothesizing that the magnitude of stressful life event change is predisposing to and predictive of future illnesses and/or complaints. Although there is a considerable and still growing body of evidence to support the contributions of life event stress to subsequent psychological and physical disorder, the resultant explosion of research in this area has highlighted the complexity of the issues in the diathesis-stress model of illness even as researchers have continued to attempt to clarify the basic questions. The focus of the present study was to improve prediction of physical and psychological complaints subsequent to life event stress by more comprehensive measurement of

life event characteristics and also by assessment of factors related to the person experiencing the events in the stress-illness process. It was hypothesized that inclusion of these event-related and person-related factors would illuminate some of the relationships in the life stress process as well as improve prediction.

Event-Related Predictor Variables

The results of the present study suggest that the simple counting of life event occurrence is an effective and parsimonious predictor of subsequent psychological and physical complaints as compared to more elaborate methods for measuring life events. Although each of the idiographic subscales of the experimental version of the Life Experiences Survey showed strong associations with future complaints, their addition did not improve the predictive model based on event occurrence alone. The strength of relationship found in this study between life event measures and illness measures is comparable to or stronger than that reported in similar research (Rabkin & Streuning, 1978).

It would appear that the current study would support proponents of the position that both positive and negative life change contribute to illness (e.g., Coppel, 1980; Petrich & Holmes, 1977; Sarason et al., 1980) as opposed to those who hold that only negative events

contribute to the stress-illness process (Mueller et al., 1978; Sandler & Lakey, 1982) or only undesirable, uncontrolled events (McFarlane, Norman, & Streiner, 1983). In the present study, a strong relationship was found beteen the occurrence of life events, both positive and negative, and future psychological and physical complaints. The association of complaints with the LES subscale for the total number of events (LIFE 1) was stronger than this association with the LES subscale for desirability of events (LIFE 2). One recent study (Matheny & Cupp, 1982) might elucidate some of the confusion as to what constitutes a stressful event (e.g., all events versus only undesirable events). Matheny & Cupp used a succession of adjusted life change scores for each subject to reflect the moderator variables of control, anticipation, and desirability for every item the subject checked on the Schedule of Recent Experiences. They then established a baseline correlation between the total unadjusted SRE score and their illness measure for the total group and for men and women separately. Correlation coefficients were then computed for all conditions reflecting the presence of positive and negative moderator variables, singly and in combination. Life events with negative moderator variables were those events that subjects considered to be undesirable, uncontrollable or unanticipated; whereas,

life events with positive moderator variables were those events that were considered to be desirable, controllable, or anticipated. Only a few of the 48 correlations they obtained will be discussed for the sake of brevity.

All life events with negative moderator variables, considered singly, were significantly correlated with illness for men and women separately and as a group. The three types of positively moderated events had very low correlations with illnesses for men and women separately and as a group with one exception: The occurrence of desirable events was significantly correlated with illnesses for women. Of the negatively moderated events, only events beyond control had a correlation with symptoms significantly higher than baseline for all groups. The correlation with symptoms of events that were considered undesirable was significantly beyond baseline only for men, whereas the correlation with symptoms events that were anticipated was beyond baseline only for women. Combinations of negative variables also produced mixed results. Combinations of positive and negative moderators produced results similar to baseline except for events that were considered desirable but beyond control; the correlation of these events with symptoms was significantly higher for women. All in all these results suggest that although moderator variables

such as perceived control, desirability, and anticipation can affect the stress-illness relationship, the effect is not consistent for all variables for all groups.

It seems likely that use of life event instruments which include assessment of several moderator variables, such as the experimental version of the LES, may be premature. Given the continued lack of consistent findings between studies and, even within studies, as to the effect of anticipation, control, and desirability on life events, the use of a simple count of event occurrence seems best for most research investigating the life stress-illness process.

Person-related Moderator Variables

A detailed examination of the relationships found between the predictor and criterion variables in this study has resulted in some conclusions that are inconsistent with the traditional models of the life stress-illness process. Regardless of the increasing complexity of the stress models in recent years (Dohrenwend & Dohrenwend, 1981), examination of the process has typically been based on the assumption of psychosocial stressors such as life events in the initial or causal position, event-related, person-related, or environmental variables in medial or moderating positions, and symptoms, complaints, or illness in the terminal position. Results of the present study along with other current research suggest that it may be

important to "see the relationships among variables as loops and circles and less as straight lines" (Leavy, 1983, p. 18).

Somatization. When one examines the current data, the power of life events to predict future complaints is severely reduced once the factor of somatization is considered. It would seem that the fundamental nature of the stress-illness model may need to be changed. Given that the response tendencies assessed by the MMPI Hypochondriasis Scale (HS) reflect a personality or response style that is stable over time (Greene, 1980), the causal implications of these tendencies are more sustained than those of life changes, which are temporally bound, and by definition, require adaptive change (Cooke & Rousseau, 1983). Thus, it might be more appropriate to view enduring personality or response style factors such as somatization as independent variables, complaints as dependent variables, and environmental forces such as life events as moderating variables. Persons with certain cognitive, physiological, emotional, and/or behavioral characteristics may be expected to be more susceptible to psychological or physical complaints, especially when adaptive behavior is required, because of a predisposition toward dysfunctional reactions and responses.

Since the other measure of somatization, the Hyste-

ria Scale (HY) of the MMPI, was so highly correlated with HS, it seems likely that the tendencies involved in these predispositions toward complaints following life events reflects the joint contribution of HS and HY traits. There would appear to be four factors that may be involved in this predisposition: 1) a tendency to focus on the somatic components of depression, anxiety, and generalized emotional arousal (Barsky, 1979; Katon, Kleinman, & Rosen, 1982) which are the major emotional reactions that have been associated with stressful situations (Thoits, 1984) and to deny the problematic nature of stressful situations (Carson, 1969; Greene, 1980); 2) a tendency to attend to and amplify peripheral sensations or perceptual components of symptoms that commonly occur and most people ignore (Barsky, 1979); 3) empirical evidence that some hypochondriacal persons display elevated basal levels of physiological arousal and heightened perceptual sensitivity to their own physiological state, leading to the tendency to perceive more bodily sensations (Hanback & Revelle, 1978); and 4) the finding that persons with HS & HY elevation show physiological hyperresponsivity demonstrated by elevated and/or prolonged stress responses (Vickers, 1983). Mechanic (1983) has reviewed data from a variety of research areas that suggests that attention to inner feelings and bodily changes increases awareness and

potency of distress and prevalence of reported symptoms.

Most of the literature on somatization relates to the first factor and has been frequently interpreted as representing a defense mechanism in that focussing on physical sensations could serve to distract one from painful emotions or attending to the actual problem causing distress (Barsky, 1979). It has been suggested that this tendency is due to an overemphasis on body functions and a history of illness during childhood (Coleman, 1976). Studies have also demonstrated both familial and cultural constraints and norms regarding the way in which distress is to be expressed. It is only in the modern Western world that a psychologically sophisticated vocabulary for verbalizing emotions has been developed. Even so, Western society still places fewer negative connotations on physical disability than emotional disability (Katon et al., 1982). Although subjects in the current study did endorse psychological complaints as well as physical complaints, as would be expected of a more educated group, a stronger relationship was still seen between the predictor variables of life event and somatization factors and the criterion of physical complaints.

A final issue needs to be considered in interpreting the strong relationship between somatization and future complaints. Factor analyses of the HS Scale have consis-

tently identified common factors labelled "poor physical health" (Comrey, 1957; O'Conner & Stefic 1959) and subjects with hypochondriacal predispositions have been found to report more current health complaints, disorders, and symptoms (Smith, Snyder, & Perkins, 1983). Since the current study did not control for emotional well-being and physical health status at the time of the initial data collection, it is possible that the associations between somatization factors and complaints primarily reflects stable differences in physical health between individuals. Recent longitudinal studies that have considered temporal relationships between physical and/or mental health, life events, and subsequent health have found that the best predictor of present health is prior health and that the inclusion of prior health in the stress-illness model reduces the association between life events and future illnesses, disorders, or complaints (Billings & Moos, 1982; Kobasa, Maddi, & Courington, 1981; McFarlane et al., 1983; Williams, Ware, & Donald, 1981). Studies (Billings & Moos, 1982; McFarlane et al., 1983; Nelson & Cohen, 1983) have also found life stress scores to be reliable over time, with past life stress being a good predictor of future life stress. McFarlane and his associates, in a methodologically sophisticated longitudinal study, found both life stress and health status to be stable over

time. In this study, the effects of life events on changes in health were significant but much lower than reported in previous cross-sectional research. They also found a reciprocal effect in that physical symptoms and emotional distress lead to the experience of stressful events. They suggest that some other factor or factors, such as personality or constitution, may lead to stressful events, emotional distress, and poorer health. The findings from the present study that HS and HY are not only strongly associated with future complaints but also with life events, suggest the possibility that these scales tap a characteristic of persons that fit an hypothesized underlying factor, like that proposed by McFarlane et al. (1983), in the stress-illness process. The findings of elevated basal physiological arousal and heightened perceptual sensitivity (Hanback & Revelle, 1978), along with physiological hyperresponsivity (Vickers, 1983) demonstrated in hypochondriacal/somatisizing persons suggests a constitutional or acquired physiological bias towards the experience of emotional and physical complaints which warrant further investigation. The association of these personality traits with the occurrence of stressful events also needs to be examined further, preferably in a longitudinal design.

Locus of Control

In the present study, locus of control was found to be associated with the future occurrence of both psychological and physical complaints, with greater external attribution of control being associated with an increased frequency of complaints. This relationship was independent of the occurrence of life events, with the correlations of locus of control and life events factors approaching zero. Although some studies have reported locus of control to have a moderating effect on the life event-illness process (Johnson & Sarason, 1978; Sandler & Lakey, 1982), others (e.g., Nelson & Cohen, 1983) have found the relationship of control to future symptoms to be independent of the occurrence of stressful life events. Locus of control beliefs were unrelated to perceived control over life events, a finding that has been reported previously (Nelson & Cohen, 1983; Sandler & Lakey, 1982).

Social Supports. None of the social support measures were associated with future complaints, occurrence of stress, somatization, or locus of control. It is possible that the presence of social support of some type might have provided a buffering effect that was not detectable due to the research design or analysis itself. For example, social support might have demonstrated a protective effect only at high levels of life stress. In

order to assess the presence of this type of conditional effect, a statistical interaction term is typically used (Cleary & Kessler, 1982). However, due to the sample size and the number of independent variables being considered in the current study, the interaction terms necessary to assess a possible buffering effect for any of the social support measures could not be included in the regression analyses generated for this study. It should be noted that there is still a great deal of controversy over the conceptual, methodological, and theoretical issues (Bruhn & Phillips, 1983; Cleary and Kessler, 1982; Thoits, 1982) involved in studying the effects of social supports in the life stress process. Regardless of the way in which social supports are studied, however, a growing number of studies have reported finding no buffering effects, only direct effects of social support on outcomes, and others have found direct as well as buffering effects (e.g., Andrews et al., 1978; Leavy, 1983; Lin et al., 1979; Thoits, 1982; Williams et al., 1981). Therefore, it is somewhat puzzling that no direct effects were found in the present study.

The interview schedule for Social Interaction (ISSI) was chosen to measure the variable of social support since it had the best theoretical basis as well as empirical validity and reliability of any measure found.

A recent survey (Bruhn & Phillips, 1984) of fourteen techniques for measuring social supports (not including the ISSI) that have been used previously noted that only four had any reported evidence of reliability and validity. None of the techniques mentioned had been as extensively tested in their development as was the ISSI (Henderson, 1981). Therefore, it seems unlikely that the failure to find any effect was due to the instrument itself. The subjects in the current study were within the expected limits for a "normal" population on the ISSI, although the measures of adequacy of support for both attachment and social integration were at the low end of the expected range. However, it should be noted that the availability of social support scores (AVAT and AVSI) for this sample were much closer to the means Henderson, et al. (1981) found (AVAT $\bar{X}$ = 6.10, ADAT $\bar{X}$ = 75.2, AVSI $\bar{X}$ = 9.69, ADSI $\bar{X}$ = 12.41) for the age group 18 - 24 than were the perceived adequacy of social support scores, suggesting that the sample had a comparable social support group in terms of quantity but less so in terms of perceived quality of support. However, a lower perception of adequacy should increase the likelihood of finding effects. Henderson and his associates found the relationship between the perception of social relationships as inadequate and the subsequent onset of psychological symptoms to be much stronger under

adversity but present even without adversity (i.e., both direct and buffering effects). Henderson did note, however, that the role of protective influence from the immediate social environment can only be small compared to the large effects of long-term intrapersonal or constitutional factors.

Another reasonable explanation for the lack of findings is the possibility that this instrument, having been constructed and normed with a general population, was not adequate to assess the social support systems of this specific population. Several of the subjects in the study did comment on the fact that the ISSI only counts members of one's support network that live within 30 miles of one's current residence. Thus, strong family and friendship ties in the home towns of the student were not considered. This may have distorted the findings since a student who grew up 40 miles away from LSU and went home every weekend but had only a few friends on campus might appear to have a less available and adequate social support network than a student from another country who had a slightly larger group of friends on campus.

The "Best" Model: Research and Clinical Implications

In this study, the combination of the HS scale of the MMPI, the change and desirability subscales of the LES (experimental), and the I-E score combined to form

the most parsimonious and powerful set of predictors for future complaints. All of these factors demonstrate direct relationship with future complaints and the effects seem to be additive. Although the effects of stressful events examined alone were actually greater than that reported in many other studies (Rabkin & Struening, 1976), they added only a little to the predictive model, as did dispositional beliefs about control.

This model and the findings which resulted in its derivation have several implications for future research. It would appear that the more the stress-illness process is studied, the more complex it is revealed to be. In order to adequately assess the stable, reciprocal, and interactive patterns that have been identified in this process thus far, prospective, longitudinal, and even life-span research strategies will be needed. The stable as well as the sequential aspects of this process require examination across time and setting.

The current findings of the importance of somatisizing or hypochondriacal traits in the stress-illness process along with the research already cited which suggest increased basal arousal and hyperresponsivity of persons with these traits suggests a possible mechanism for the development of psychosomatic disorders. Studies have shown that when stressors elicit emotional arousal, a complex physiological reaction

ensues (Frankenhauser, 1980; Levi, 1975; Mason 1975b, 1975c). Prolonged, intense, and/or frequent emotional reactions to stressful situations, through activation of the immunitary, endocrine, and autonomic nervous systems, can result in weakened biological defenses against disease (Pancheri, 1979) or can initiate structural and/or physiolgical dysfunctions directly (Miller, 1980; Rogers et al., 1979). The intensity of the response to stress is determined by cognitive appraisal and personality characteristics or response styles as well as actual stimulus parameters. Finally, the characteristics of the psychobiological response (i.e., emotion) are conditioned by genetic factors, learning, and biological changes (Pancheri, 1979) as well as affected by situational cues, current physiological state, cognitive interpretation, and ongoing behavior (Thoits, 1984). Thus a complex of biological interactions occur in which enduring personality characteristics or response styles play a relevant and, perhaps, at times, determinant role.

The implications of these findings for clinical practice are rather heartening, as the response styles or behavioral patterns which were found to be associated with vulnerability to illness are amenable to psychological intervention (Harris, 1983; Lachman, 1972; Nicholi, 1978; Wittkower & Warnes, 1977). Future research investigating the presence of physiological

differences associated with the personality trait of somatization will be pertinent to the planning of intervention strategies. If there are maladaptive physiological patterns associated with vulnerability, techniques for teaching people to reduce their physiological activation level, such as progressive muscle relaxation, autogenic training, and biofeedback will need to be part of any intervention strategy for vulnerable persons. Two extensive reviews (Begnum & Begnum, 1983; Tarler-Benlolo, 1978) of relaxation techniques as therapeutic interventions have concluded that these techniques represented an easily taught, effective coping strategy which may prevent and treat stress-related disorders.

Footnotes

[1]This is an unpublished, revised version of the LES (Sarason, Johnson, & Siegel, 1978), available from I. G. Sarason, Department of Psychology, University of Washington, Seattle, WA 98195. Used by permission.

[2]The ISSI was devised and developed by the National Health and Medical Research Council Social Psychiatry Research Unit at the Australian National University, Canberra, ACT 2600. Used by permission.

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APPENDIX I

Life Experiences Survey (LES)

The revised version of the LES is a 64-item self-report measure that allows respondents to report noteworthy events that they have experienced during the past year. The time required for self-administration may vary from 15 to 45 minutes. The first fifty-four events in the LES refer to life changes common to individuals in a wide variety of situations. Nine events are relevant primarily to student populations. Additionally, the last item provides a "blank space" to allow subjects to indicate the occurrence of any significant event they may have experienced that was not on the LES list.

This instrument is distinguished from other measures of life experiences by four major features. First, it allows the subjects to indicate whether recent life events were seen as positive or negative (i.e., desirability). Second, it permits individualized ratings of the extent to which events affected subjects' lives (i.e., change). Third, the subjects can indicate whether the events were expected to happen and if expected, with what degree of likelihood, (i.e., anticipation). And fourth, respondents can report how much control they perceived themselves to have over each event's occurrence. The last two features, ratings of anticipation and control, are the main additions to the

original LES which provided positive and negative life changes scores and individualized impact ratings. Research is currently being carried out on the revised version of the LES (Sarason, et al., 1982) but no data has been published yet. Since the new version has fifty-six items in common with the original and, still provides positive, negative, and total change scores, it is likely that the new version should be similar in terms of reliability.

For the original version, two reliability studies with a six week interval between test and retest, using positive, negative, and total life change scores, have been reported (Sarason, Johnson, & Siegel, 1978). Test-retest Pearson product-moment correlations for positive change scores were .19 (n.s.) and .53 ($p < .001$) and .88 ($p < .001$), and for total change scores were .63 ($p < .001$) and .64 ($p < .001$). In addition, to the two reliability studies, data are reported for a smaller group of subjects who took the LES on two occasions eight weeks apart as a part of another investigation. For that study reliability coefficients of .61 ($p < .05$), .72 ($p < .01$), and .82 ($p < .001$) were obtained for positive, negative, and total life change scores, respectively. Sarason and his colleagues note that test-retest reliability coefficients with this type of instrument are

likely to underestimate reliability as the time intervals allow the experiencing of more events which would be reflected at the time of retesting. Also, events for a time period equivalent to the test-retest interval, occurring at the beginning of the reporting period for the first testing, would (accurately) no longer be included in the second reporting. As some response change between the two testings would be expected and would not reflect inconsistencies in reporting, it would be inappropriate to consider the total variability in responding as error.

The reliability levels reported with this instrument seem at least adequate when compared to the results reported by Neugebauer (1981) in his review of reliability studies of various life event scales. He stated that reliability estimates reported were low to moderate in general. However, he noted that lower levels of reliability in a variable decrease the measured correlation between that variable and any other variable. According to Neugebauer, since life event scales have low to moderate reliability, this implies that the correlations reported in most life event research may actually be under-estimates of the true strength of association between event occurrence and illness onset.

Negative life change scores from the LES have been

found to relate significantly with state and trait anxiety (Sarason et al., 1978), poorer academic achievement (Knapp & Magee, 1979; Sarason et al., 1978), myocardial infarction (Pancheri et al., 1980), menstrual discomfort (Siegel, Johnson, & Sarason, 1979a), the attitudes of mothers of at-risk infants (Crnic, Greenberg, Ragozin, & Robinson, 1980), and job satisfaction (Sarason & Johnson, 1979). It has been reported that LES scores are not biased by a social desirability response set (Sarason et al., 1978). Also, experimentally induced mood states did not affect any LES scores (Siegel, Johnson, & Sarason, 1979b), suggesting that responses are not unduly influnced by the mood of the respondent.

UNIVERSITY OF WASHINGTON
SEATTLE, WASHINGTON 98195

Department of Psychology NI-25

April 21, 1983

Ms. Karen Speier
150 Alello Drive
Baton Rouge, LA 70806

Dear Ms. Speier:

You have my permission to use the Life Experiences Survey in your research. Enclosed is a copy of the LES and a reprint of an article regarding its development. Should you use the LES in your research, I would appreciate receiving a summary of your results. Best of luck.

Sincerely,

Irwin G. Sarason

Irwin G. Sarason
Professor

IGS:mec
enclosures

APPENDIX II

Interview Schedule for Social Interaction (ISSI)

The ISSI was developed as an instrument to measure social relationships, theoretically based on the concept of attachment in adults developed by Bowlby (1969-1980) and on the major "provisions" afforded by social relationships as delineated by Weiss (1974). Using a structured interview format, 52 questions explore various types of relationships and provisions of relationships (e.g., reassurance of personal worth) in terms of availability and perceived adequacy. This instrument, a full description of its development and content, usage guidelines, instructions for computing the ISSI indices, and reliability and validity studies along with results from a sophisticated two-phase (i.e., cross-sectional and longitudinal phases) epidemiological study are included in Neurosis and the Social Environment (Henderson, Byrne, & Duncan-Jones, 1981). Four main scores are calculated for each respondent:

1. AVAT - the availability of attachment, i.e., the closest interpersonal relationships, providing a sense of security.

2. ADAT - the perceived adequacy of attachment.

3. AVSI - the availability of social integration, i.e., membership in a range of relationships involving shared concerns and interests.

4. ADSI - the adequacy of social integration.

The number of ADAT questions is dependent on the number of AVAT questions responded to positively, and thus has an upper limit that varies from respondent to respondent. Thus, a supplementary score, ADAT% has been typically used instead of the ADAT score to avoid the interdependence between the AVAT and ADAT measures. ADAT % is the ADAT score expressed as a percentage of the number of AVAT questions each respondent was asked.

Reliability was assessed in terms of internal consistency on 756 subjects using the Cronbach alpha for the four main scales with the coefficients being .67, .69, .71, and .79 for AVAT, ADAT%, AVSI, and ADSI respectively. Test-retest reliability was examined using a random subsample of the 756 subjects, with fifty-one persons being re-interviewed after 18 days by different interviewers. The test-retest correlations were .76 for AVAT, .71 for ADAT%, .75 for AVSI, and .75 for ADSI. Based on methods described by Bielby & Hauser (1977), and Rogosa (1979), the stability of the social and behavioral dimensions underlying the ISSI scores at 4, 8, and 12 months here estimated. For these time periods, AVAT correlations were .88, .87, and .85; AVSI correlations were .87, .84, and .85; ADAT correlations were .80, .77, and .69; ADSI correlations were .74, .72, and .66.

Face validity of this test is adequate in that the

items effectively tap the constructs of availability and adequacy of adult attachment and social integration (Henderson, Duncan-Jones, Byrne, & Scott, 1980). First, the findings for several demographic groups where differences in social integration could be expected *a priori* were in the predicted pattern, thus demonstrating criterion-related validity. For example, persons who had arrived in Canberra in the previous six months had less availability ($p < .001$) and less perceived adequacy ($p < .001$) of social integration than people who had lived there for seven months or more. Thus, newcomers, who would be expected to have fewer social ties than longer-term residents, did so according to this test. Accuracy of self-report information on this test was assessed (Henderson et al., 1981) by having close informants respond to the questions for a random sample of the respondents ($n = 1,114$). The product-moment correlations between the matched scores were .42 for AVAT, .39 for ADAT, .59 for AVSI, and .26 for ADSI, with all being significant ($p < .01$). Validity was further assessed by consideration of the possibility of scores being affected by response style or social desirability. Two separate scores of the Crown-Marlowe Inventory (Crown & Marlowe, 1964), which assesses need for approval, and the Lie scale from the Eysenck Personality Inventory

(Eysenck & Eysenck, 1964) were correlated with the ISSI scale scores for the sample (n = 225). Using the three measures of response style in a multiple regression equation, the percentages of explained variance were 5.8 for AVAT, 8.4 for ADAT, 5.7 for AVSI, and 10.6 for ADSI, which was judged to be acceptable (Henderson et al., 1980). Thus, neither factor of response style or social desirability was an important determinant of ISSI scale scores, a finding which adds to the validity of the test.

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3.4.82

Dear Miss Sobotka,

Thank you very much for your inquiry about the ISSI. Together with its guidenotes and instruction for computing the ISSI indices, this is now published as follows:

Henderson, Scott with Byrne, D.G. and Duncan-Jones, P. (1981) Neurosis and the Social Environment. Academic Press, Sydney, New York and London.

We hope you find the instrument useful in your own research. Please let us know of any publications which ensue.

With good wishes,

Yours sincerely,

A. S. Henderson
Director

ASH:ds

APPENDIX III

Internal-External Control of Reinforcement Scale (I-E Scale)

This scale, developed by Rotter (1966), consists of 29 forced choice items, including six filler items designed to make the test purpose less obvious. This test is self-administered and usually takes 10 to 15 minutes to complete. The subject describes his own belief system by choosing between alternatives that reflect a fatalistic, external control point of view and those which indicate a belief in his or her own ability to affect and have control over the events in life. The obtained score is the total number of external choices. It has been noted that locus of control is not an encompassing trait such as competence or intelligence that relates to all facets of human endeavor. Rather, it is a form of self-appraisal of the degree to which persons view themself as having some control over specified events (Lefcourt, 1976). The concept of internal versus external control of reinforcement developed out of social learning theory (Rotter, Chances, & Phares, 1972) and described the degree to which an individual believes that reinforcements are contingent upon one's own behavior. In his review of the research on the internal-external control concept, Joe (1971)

notes that the data tend to support Rotter's contention that this concept is a generalized expectancy operating across many situations. He also reports that several studies suggest that the locus of control variable influences the strategies of individuals in confronting problem-solving and risk-taking situations.

Joe (1971) also reports that later research by other investigators on reliability for the I-E Scale has been consistent with that reported by Rotter (1966). Internal consistency estimates Rotter reported were .73 in a study of split-half reliability (n = 100), and .73 (n = 100), .70 (n = 400), and .69 (n = 1,000) in three studies using the Kuder-Richardson formula for estimating reliability. In two studies of test-retest reliability for a one-month period, reliability coefficients of .72 (n = 60) and .78 (n = 28) were reported. In a third study, using a two month interval between testings, reliability was lower r_t = .55; n = 117). Rotter suggests that this lower reliability may be partly a function of the first test being given under group conditions and the retest administered individually. Means and standard deviations for a variety of populations are reported by Rotter (1966, p.15). However, these means may not be accurate for similar populations currently. In the nine years since they were published, Rotter (1975) reports that the

mean for college students had risen from a score of 8 to somewhere between 10 and 12, depending upon the sample.

Test development, information on additional test characteristics, fuller evidence for the validity of this scale, and information on correlates of this scale are provided in the reviews by Rotter (1966), Lefcourt (1966), Joe (1971), and Rotter (1975). The most significant evidence of the criterion validity of the I-E scale comes from predicted differences in behavior of individuals classified as internal versus external or from correlations with behavioral criteria as found in a series of studies reported by Rotter (1966). A further series of studies are reported to support the discriminant validity of the I-E scale based on low correlations found with such variables as intelligence (r = -.09, n = 107; r = -.11, n = 72; -.01, n = 80) and social desirability (r = -.21, n = 306; r = -.22, n = 136; r = -.12, n = 180; r = -.29, n = 180; r = -.28, n = 113).

The internal-external variable has been investigated in research concerned with a variety of topics including attempts to control the environment, achievement, reactions to threat, risk-taking, anxiety, learning, adjustment, ethnic group and social class differences, conformity and frustration (Joe, 1971; Lefcourt, 1966,

1976). Research on locus of control as an important factor in the life event-illness onset process was cited earlier in the introduction section of this paper.

A copy of the I-E Scale is not included as this is a widely used test and therefore is readily available.

APPENDIX IV

Minnesota Multiphasic Personality Inventory (MMPI)

The MMPI is a widely used objective device for the assessment of personality characteristics. The instrument contains 550 statements covering a wide range of subject matter to which the subject can respond true, false, or cannot say. The time required for administration may vary from 45 to 90 minutes. Very little instruction and supervision are required, so group administration is quite practical (Hathaway & McKinley, 1967).

The MMPI scales were empirically developed by comparing normal groups (total n = 950) with carefully studied clinical cases (n = 800). The details of scale derivation are too extensive for brief description, but the chief criterion for acceptance of a scale was valid prediction of clinical cases as compared with the neusopsychiatric staff diagnosis, rather than statistical measures of reliability and validity. For a detailed discussion of the construction of the MMPI and the derivation of the basic scales, see Welsh and Dahlstrom (1956).

Research has indicated that short-term test-retest reliability of the MMPI is generally quite high and that a progressive decline occurs as the time interval

increases. Summarizing reliability studies on students, coefficients ranged from .81 to .96 with a one-day test-retest interval (Faschingbauer, 1973), from .62 to .92 with a one-week interval (Windle, 1955), ranged from .42 to .76 for an eight month interval (Mauger, 1972), and from .34 to .61 with a three year interval (Hathaway and Monachesi, 1963). A similar pattern of high short-term reliability with decline over time was also seen in studies using psychiatric samples (Widom, 1979).

According to Hathaway (1980), patients who obtain a high T score on a scale are ipso facto like the group from which the scale was derived, unless they tried to score high with an ulterior purpose. The latter is detectable in the MMPI's validity scales. Dahlstrom and Dahlstrom (1980) have noted that the basic scales have implications beyond prediction or description of the clinical syndromes for which they were constructed, with considerable variance in the scales reflecting predispositional features of the criterion syndrome. The work of Endicott and his associates has shown that level of elevation on a given scale has dependable ties with rated severity of disorder, and changes in elevation reflect alterations in clinical status of psychiatric patients (Endicott & Jortner, 1966; Endicott, Jortner, & Abramoff, 1969).

The MMPI has been used with a wide variety of

populations, in many settings, and for almost innumerable purposes. For discussion of its clinical and research use, see Dahlstrom and Dahlstrom (1975), Butcher and Panacheri (1976), Butcher (1979), and Newmark (1979). After reviewing the use of MMPI in the study of psychosomatics, Pancheri (1979), concludes that the most interesting applications in the next few years will probably be in the area of identification of links between particular emotional patterns and biological alterations preceding disease. He suggested that this would make timely psychotherapeutic intervention possible.

A copy of the MMPI is not included as this is a widely used test and therefore is readily available.

APPENDIX V

SCL-90-R.

The SCL-90-R is a 90-item self-report symptom inventory developed by the Clinical Psychometrics Research unit of Johns Hopkins University School of Medicine (Derogatis, Lipman, & Covi, 1973; Derogatis, Rickels, & Rock, 1976). Each item is typically rated on a 5-point scale indicating how much the respondent was bothered by a symptom, ranging from not at all (0) to extremely (5). The SCL-90-R reflects psychopathology in terms of nine primary symptom dimensions and three global distress indices. The nine symptom dimensions are Somatization (SOM), Obsessive Compulsive (OBS), Interpersonal Sensitivity (INT), Depression (DEP), Anxiety (ANX), Hostility (HOS), Phobic Anxiety (PHOB), Paranoid Ideation (PAR), and Psychoticism (PSY). The three global indices, although correlated, are summary measures of psychological disorders that have been shown to measure distinct aspects of psychopathology (Derogatis, Yevzeroff, & Wittelsberger, 1975). The General Severity Index (GSI) combines information on numbers of symptoms and intensity of distress, while the Positive Symptom Total (PSI) reflects numbers of symptoms, and the Positive Symptom Distress Index (PSDI) is a pure intensity measure, adjusted for number of

symptoms present. The SCL-90-R is a measure of current psychological symptom status and not a measure of personality characteristics (Derogatis, 1977).

The standard time set given with the SCL-90-R is to report presence of symptoms for the past "7 days including today." It is designed with a flexible time window so that evaluations over other time periods can be made. However, informal studies by the Clinical Psychometrics Research Unit (Derogatis, 1977) have shown that time referents longers than 14 days begin to introduce distortions as a result of memory processes as well as natural variability of symptoms over time. This test can be administered in a group or individually. Under usual circumstances, it requires 10-20 minutes to complete.

Internal consistency measures for the 9 dimensions were calculated (Derogatis et al., 1976) using coefficient alpha, a multipoint variation of the Kuder-Richardson 20 formula (n = 219 "symptomatic volunteers"). Coefficients ranged from a low of .77 for Psychoticism to a high of .90 for Depression. Test-retest coefficients were obtained from psychiatric out-patients (n = 94) during initial evaluation, and again one week later prior to their first therapy appointment. Coefficients ranged from .78 for Hostility to .90 for

Phobic Anxiety. Edwards, Yarvis, Mueller, Zingale & Wagman (1978) reported an overall coefficient alpha of .95 and a series of test-retest coefficients for different time periods with a range of .81 to .94, and a mean of .86.

In a study of concurrent criterion-oriented validity, the SCL-90-R dimension scores were contrasted with MMPI scores (Derogatis et al., 1976). The sample consisted of 119 symptomatic volunteers and the MMPI was scored for Wiggins (1969) content scales and Tryon (1966) cluster scales as well as the usual clinical scales. Correlations reported between MMPI and SCL-90 scales ranged from .40 to .75, with a mean correlation of .62 reflecting a high degree of convergent validity (Derogatis, 1977). In a similar validation study, Bolelоucky and Horvath (1974) correlated the SCL-90-R symptom dimensions with those of the Middlesex Hospital Questionnaire. With a mixed sample of 130 subjects, correlations between equivalent dimensions ranged from .36 for the Phobic Anxiety Scales to .92 for the Global Severity Index and the MHQ Global Score. A large sample ($\underline{n}$ = 1,002) factor analytic study supported the construct validity of the instrument in that the dimensional constructs hypothesized to comprise the scale were empirically confirmed (Derogatis & Cleary, 1977).

The SCL-90-R has proven sensitive to change in a broad variety of clinical and medical contexts. Research on depression has revealed it to be very sensitive to the presence and alteration of depressive disorders (Brown, Sweeney, & Schwartz, 1979; Weissman, Scholomskas, Pottenger, Prusoff, & Locke, 1977; Prusoff, Weissman, Klerman, & Rounsaville, 1980). The SCL-90-R has demonstrated high and consistent sensitivity to change in psychopharmacologic research (e.g., Ravaris, Robinson, Ives, Wies, & Bartlett, 1980). The SCL-90-R has been utilized in a number of medical contexts such as assessment of pre-post treatment changes in alcoholic couples (Steinglass, 1979), relationship of coping style to survival time with metastatic breast cancer (Derogatis, Abeloff, & Melisaratos, 1979), and assessment of biofeedback-induced changes in chronic pain patients (Hendler, Derogatis, Avella, & Long, 1977). The SCL-90-R has been shown to be sensitive to stress-related changes in a group of adults who had recently experienced the death of a parent (Horowitz, et al., 1981).

A copy of the SCL-90-R is not included as this is a widely used test and therefore is readily available.

APPENDIX VI

Wahler Physical Symptoms Inventory (WPSI)

The WPSI is a self-report measure of physical complaints with items selected to denote only malfunctions of basic somatic systems or complaints about physical states or sensations. It typically requires five to ten minutes to complete. In the standard administration, subjects respond to items as to how often one is bothered by a list of 42 physical troubles on a 6 point scale ranging from 0 (almost never) to 5 (nearly every day). The obtained score is the sum of the ratings divided by the total number of items after the number of items omitted or given more than one score are subtracted (i.e., score = sum/(42 - # omitted or incorrectly scored). No subscales are given. The total score is compared with the test norms, given in terms of deciles for males and females. It is interpreted by examination of the pattern of responses as well as the obtained decile. For example, high total scores resultant from extreme scores on a few related complaints are most often due to a serious and/or chronic illness. A 5-year study found that WPSI forms completed at home did not differ significantly in terms of means or variance from forms completed in a clinic or office (Wahler, 1973).

Internal consistency of the overall test was

established using the KR20 for scores of four patient groups (total n = 251), four university student groups (total n = 245), one rehabilitation group (n = 70), and a group seeking disability compensation (n = 97). KR20 values ranged from .88 to .94, with a mean of .90, indicating that the internal consistency of the WPSI is not only high but consistently so over diverse groups. Test-retest reliability was assessed with three groups (two student groups and one group of chronic schizophrenics) over periods ranging from one day to thirteen weeks. Coefficients of stability were high initially and declined over time. For example, with a one day interval between testings for a group of college students (n = 120) the coefficient was .94 and for a psychiatric group (n = 60) it was .64 for a thirteen week test-retest interval.

Concurrent validity was assessed by using comparison groups with different levels of expected symptomatology (i.e., health college students, psychiatric patients, disability claimants, and rehabilitation patients.) All patient groups scored significantly higher than students and the disability claimants scored significantly higher than other patient groups as was predicted.

A copy of the WPSI is not included as this is a widely used test and therefore is readily available.

VITA

Karen Rinardo Speier was born on August 19, 1947 in New Orleans, Louisiana. She graduated from the University of New Orleans in 1969 with a B.A. degree in psychology. She received an M.S. degree in psychology from the University of New Orleans in 1974.

EXAMINATION AND THESIS REPORT

Candidate: Karen Rinardo Speier

Major Field: Clinical Psychology

Title of Thesis: Symptoms in Relationship to Life Events

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

December 17, 1984